VOLUME 18 NUMBER 5

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NOVEMBER, 1933

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, $6.00

Entered as Second-Class Matter, January 14, 1925, at the Postoffice at Chicago, Illinois, Under the Act of March 3, 1879. Acceptance for mailing at special rate provided for in Section 1103, Act of October 3, 1917, authorized January 22, 1925.

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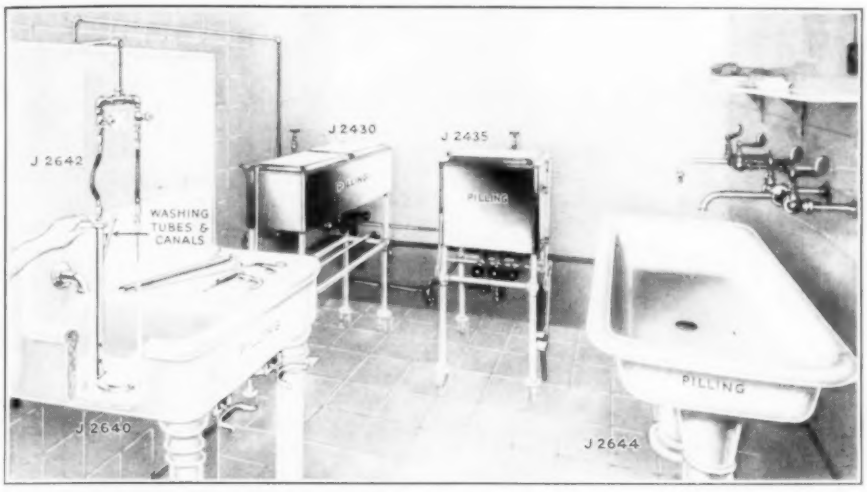

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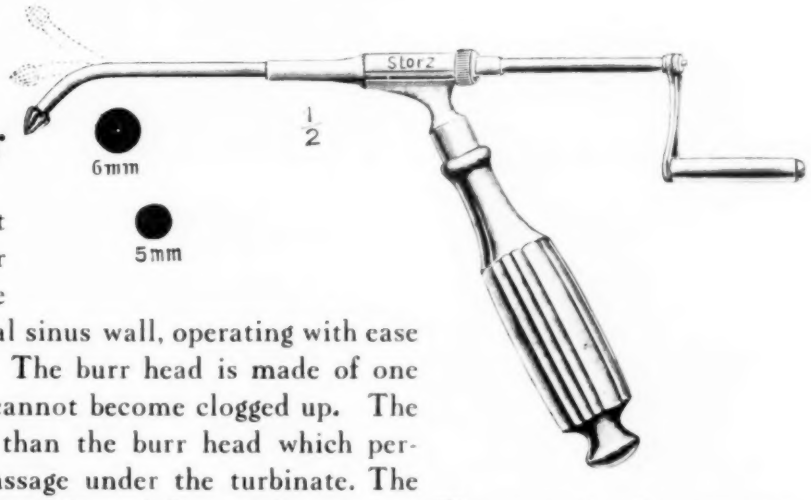

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INVOLVEMENT OF THE ESOPHAGUS IN ACUTE AND IN CHRONIC INFECTION

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BOSTON

About three years ago I ended a long search by proving to my satisfaction, and perhaps to the satisfaction of others, that cardiospasm is due to fibrosis of the terminal portion of the esophagus caused by infection from contiguous organs. I wish now to give instances of infection of the esophagus itself, in other words, of infection from within. I shall show that infection of the esophagus may occur in acute disease; for example, in pneumonia. These two subjects are quite academic and smell of the laboratory. They constitute the first part of the paper. The concluding portion deals with the fluoroscopic examination of the esophagus and the passage of the barium bag. This I shall make as practical as I can. In the discussion of infection of the esophagus, I shall take up the question of the glands as the favorite point of entrance.

THE MATERIAL

The material for the paper consists of one hundred autopsy specimens of the esophagus. Of these, about seventy-five have so far been sectioned. The macroscopic and microscopic observations furnish the basis of the first half of the paper.

The study of my present series of cases shows that fibrosis of the esophagus is fairly common, both in the subepithelial tissues and in the muscular layers. As I just said, I first proved that fibrosis can be due to infection from without. I have found that it can start in the esophagus itself, especially in the subepithelial connective tissue, and extend to the muscular layers. When it pursues this course, the circular layer is involved first, and when only one muscular layer is involved, it is generally the circular. Fibrosis seems to attack involuntary muscle first. This means that it usually begins in the lower half of the esophagus.

Acute infection arising within the esophagus occurs either as superficial ulceration or as edema of the submucosa and infiltration of the

Chairman's address read before the Section on Otolaryngology, Otology and Rhinology at the Eighty-Fourth Annual Session of the American Medical Association, Milwaukee, June 14, 1933.

connective tissue. The presence of polymorphonuclear leukocytes is necessary to substantiate the existence of acute infection. I give at least three instances in which the submucosa is fully as thick as the two muscular layers together, the muscular layers not being involved in the infection (figs. 1, 2 and 3).

Chronic infection of the esophagus is shown by a generalized infiltration with small round lymphocytes and plasma cells and by an increase in the subepithelial connective tissue, or by an increase in the connective tissue throughout the esophagus and in the periesophageal tissues. The lymphocytes often form a ribbon-like band under the epithelium. They collect in the glands and especially about

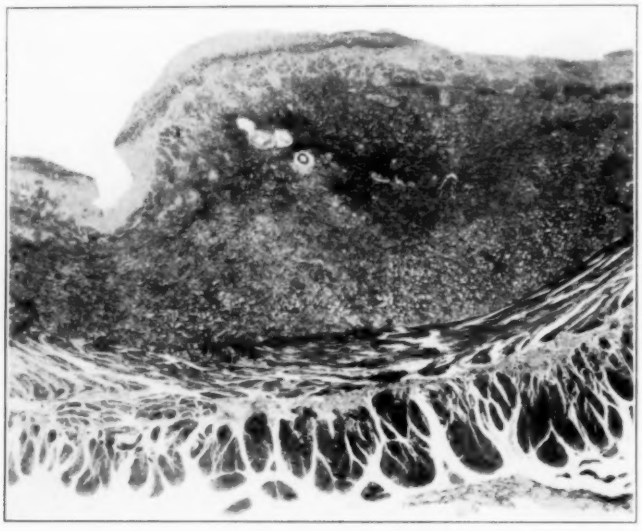


Fig. 1.—The section shows fibrinous exudate and infection in the subepithelial connective tissue. The width of the exudate equals the combined width of the circular and longitudinal muscular layers.

the gland ducts. In one instance, the collections in the glands resembled solitary follicles. In chronic infection, the number of solitary follicles under the epithelium greatly increases (figs. 4, 5 and 19).

I believe that the glands play a large part in infecting the esophagus from within, especially the superficial glands. These are almost beneath the epithelium. It has been observed that in the upper part of the esophagus the superficial glands are often found to be eroded, and that they give the appearance of shallow ulcers. It is not uncommon also to find large solitary follicles over which the epithelium is lacking, looking almost as though they had pushed the epithelium off by their great size.

The surface epithelium and the epithelium of the gland ducts seem to have a great attraction for the small lymphocytes. The cells of the glands attract them also, but to a less extent. When the lymphocytes are numerous under the surface epithelium, they swarm through the basement membrane at various points, and are seen in numbers in the epithelium. At the places where they go through, the basement membrane is broken up and the lower cells of the epithelium take on a confused arrangement strikingly like the beginning of malignant disease (fig. 6).

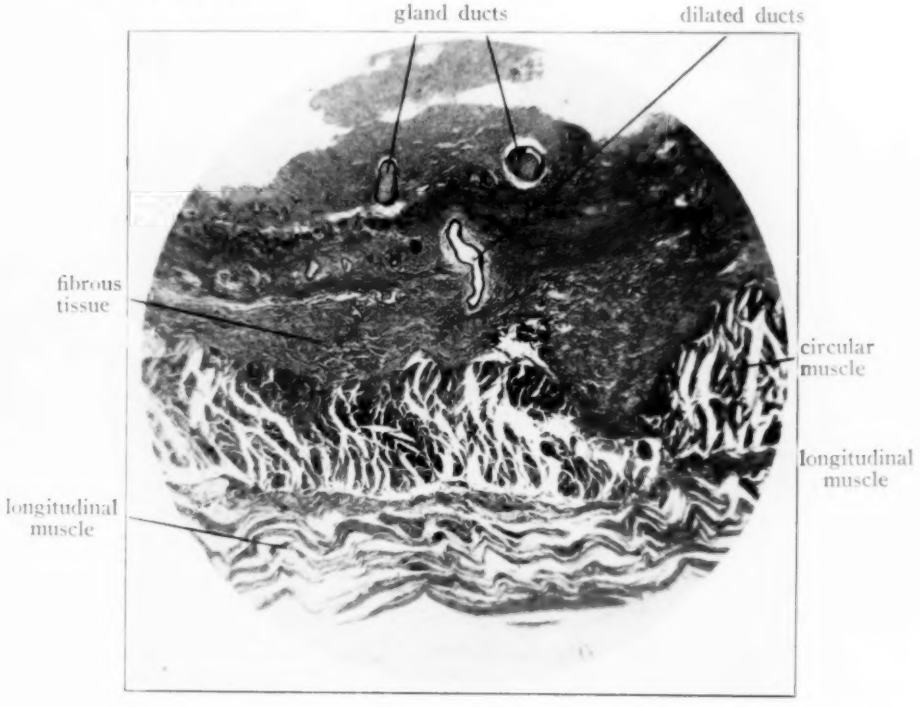


Fig. 2.—The photomicrograph shows that the epithelium is lacking. There is a subepithelial thickening due to a deposit of fibrin. Two gland ducts show hypertrophy of the epithelium which occludes one and almost occludes the other.

The epithelium of the esophagus, since there are about 16 inches (40.64 cm.) of it and because it can be followed so easily, seems to be an ideal structure in which to trace the beginning cellular changes in malignant disease.

The last few sentences are a bit of a digression. They are dangerous to put on paper because one is always accused of slipping (if not of something worse) if one discusses the early changes in cancer.

In connection with acute infection of the esophagus, it is not uncommon to have an exudate form over the site of the infected area. A case of general peritonitis to be mentioned later is a case in point.

Figure 7 from this case shows the esophagus to be spattered with numerous flakes of exudate which cover ulcerated areas. The presence of the exudate over the ulcerations would, in the healing of the ulcers, make the chances of the formation of a web or a stricture much greater.

Fig. 3.—The photomicrograph shows that the epithelium is lacking. In the subepithelial tissue the fibrin has in places turned to fibrous tissue.

Having digressed a bit, I shall anticipate a little by saying that I am especially pleased to get evidence that the esophagus can be infected from within, because years ago, in speaking of the lower end of the esophagus, I made the statement that it would require but a slight amount of infection or trauma to glue the vertical folds together and start obstruction. I shall not review the anatomic conditions at the lower end of the esophagus which favor obstruction there, except to remind one that in the resting state the vertical folds are practically in apposition.

INFECTION OF THE ESOPHAGUS FROM WITHOUT

The following two cases illustrate fibrosis of the terminal portion due to infection from without.

Fig. 4.—The photomicrograph shows a ribbon-like infiltration of small round lymphocytes under the epithelium. This indicates chronic infection.

Fig. 5.—The section shows dilated gland ducts. The glands are infiltrated with lymphocytes. The muscularis mucosa has nearly disappeared. The esophagus shows chronic infection.

Case 1.—F. O. A., a white man, aged 63, had a psychosis due to somatic disease.

He had an old (fifteen years) peptic ulcer for which posterior gastrojejunostomy was performed. He began to show signs and symptoms of obstruction and (two weeks prior to death) was again operated on, cecostomy being performed.

The symptoms and signs of peritonitis and further obstruction then developed, and he died.

Autopsy Observations.—Autopsy was performed ten hours after death. It showed the old posterior gastrojejunostomy (the peptic ulcer was not found),

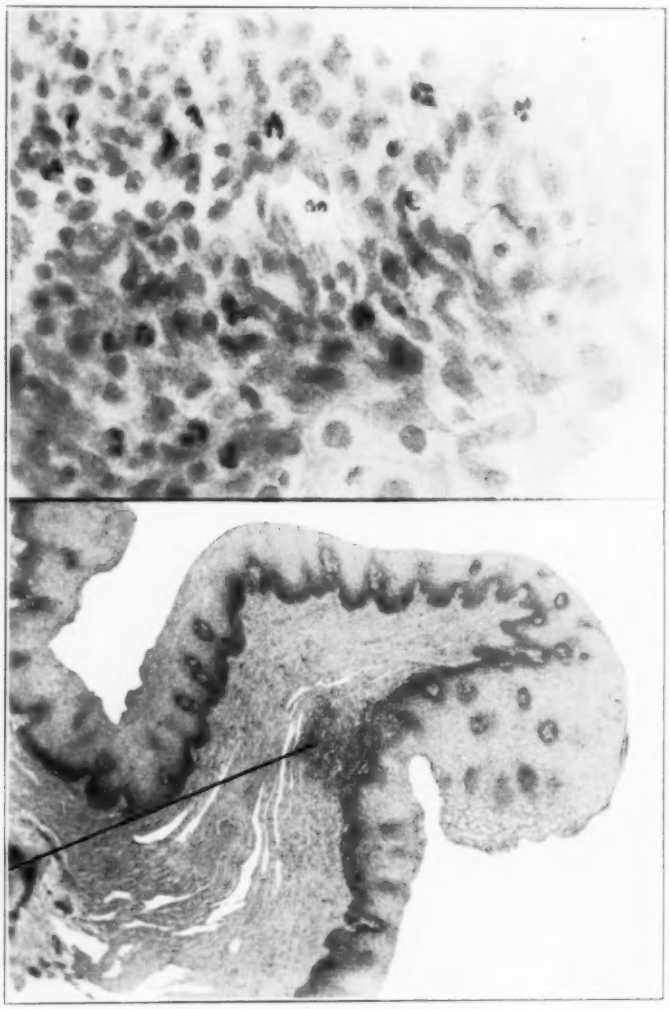


Fig. 6.—The lower photomicrograph shows an isolated group of lymphocytes under the epithelium. The basement membrane is broken at this point. The upper slide is a high power view of the epithelium over the group of lymphocytes. There are polymorphonuclear leukocytes in the epithelium. Other slides showed necrotic areas with bacteria in them.

with ulceration, hemorrhage and perforation with obstruction. The following changes were noted: the recent cecostomy with a localized abscess, multiple intestinal adhesions, mitral, aortic and myocardial fibrosis, coronary sclerosis, healed

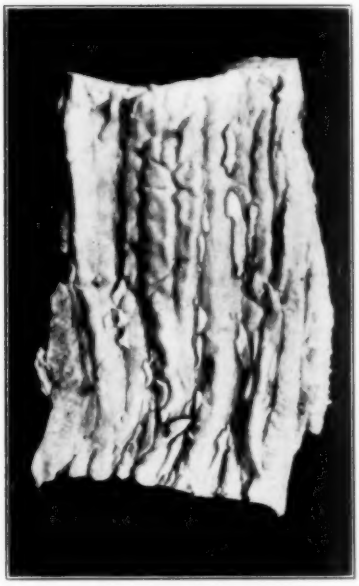


Fig. 7.—The photograph shows many patches of exudate.

Fig. 8.—The photograph shows a fibrous narrowing at the lower end of the esophagus. Above this, for one half of the length of the specimen, the muscles of the esophagus are much thickened.

apical tuberculosis, healed perihepatitis, chronic splenitis, arteriosclerosis with nephritis and a diverticulum of the urinary bladder (5 cm. in diameter).

The anterior surface of the pancreas was adherent to the posterior wall of the stomach, partially sealing the perforating ulcer.

Macroscopic Examination.—The lower end of the esophagus at the angle where the esophagus joins the stomach was markedly thickened and fibrosed. The cardiac opening was narrow and puckered into a fibrous ring. For about 4 inches (10 cm.) above the narrowing, the esophageal musculature was thickened. Above the thickened portion, the esophagus was only half as thick, that is, about 2 mm.

There was fibrosis of the cardia, probably due to infection from the previous ulcer of the stomach.

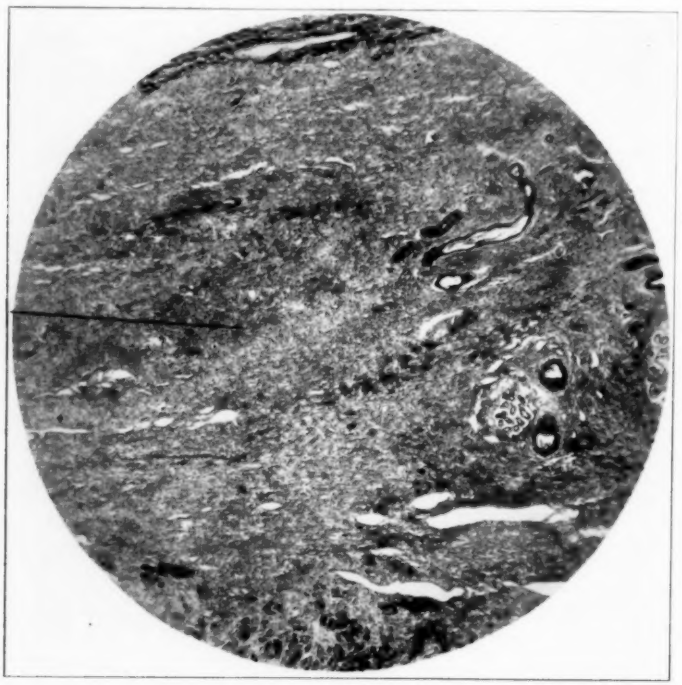


Fig. 9.—This section is taken from the fibrous constriction at the lower end of the esophagus. It shows almost no muscular tissue.

Microscopic Examination.—This showed extreme fibrosis at the junction of the esophagus and stomach. This, of course, is a marked case. Lesser degrees, in fact, all degrees, of fibrosis occur.

The slides showed dilated and infected glands, and the mucosa of the esophagus in one place showed a definite abscess with marked infection in and about it (figs. 8, 9, 10 and 11).

Case 2.—A woman had fibrosis of the terminal portion of the esophagus for at least twelve years. Once during that period I managed to dilate the narrowing sufficiently for her to get along in some comfort. She promised me her esophagus when she died, and she kept her word, or rather her relatives did.

Fig. 10.—The photomicrograph shows dilated glands in the esophagus with marked infection. The high power view showed a definite abscess in the mucosa with marked infection throughout the whole area.

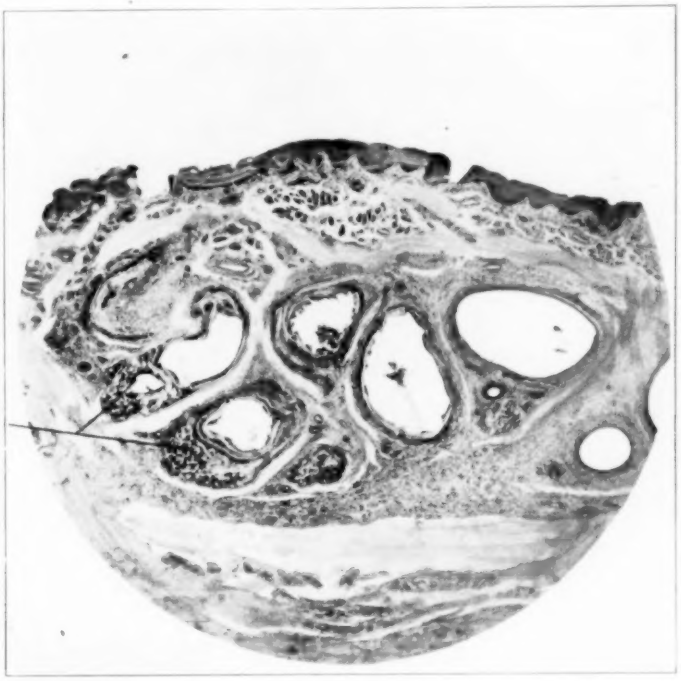


Fig. 11.—The section shows dilated gland ducts and chronic infection of the glands.

Macroscopic Examination.—The lower half of the esophagus was dilated so that it looked like a second stomach.

Microscopic Examination.—The microscopic sections showed that the part of the esophagus in the crural canal had little remaining muscular tissue, most of the tissue being fibrous. The liver was small and scarred. The gallbladder was thickened and infected, and the terminal portion of the esophagus was surrounded by stout adhesions which sprang from the sheath of the gallbladder. The disintegration of the muscular tissue extended to the fundus of the stomach. In this case, the esophagus had practically given up the ghost as an esophagus.

As chronic infection and fibrosis of the terminal portion of the esophagus from infection from without is an old subject on which I have written a good deal, I shall stop with these two rather extreme

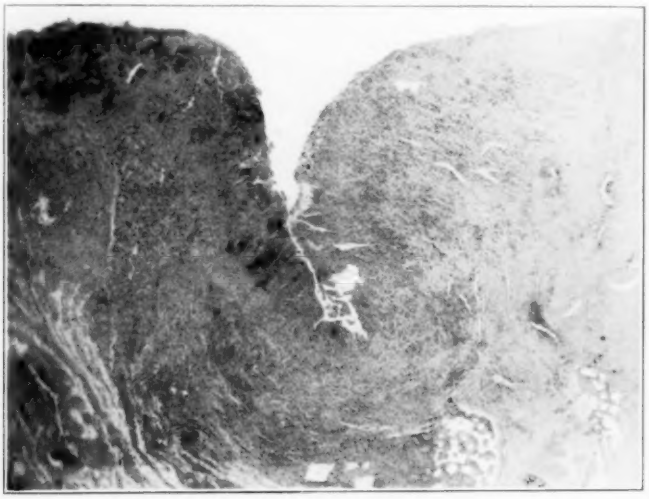


Fig. 12.—The epithelium of the esophagus is lost. The subepithelial tissues are thickened and infiltrated with polymorphonuclear leukocytes. There are many clumps of bacteria.

examples and go on to a new subject, namely, involvement of the esophagus in acute disease.

ACUTE INFECTION OR INFECTION OF THE ESOPHAGUS FROM WITHIN

The following cases show that in infections of the blood stream the esophagus, like the other organs of the body, can be involved. The first case is one of cavernous sinus thrombosis in a boy (J. B.), aged 5, in whom pneumonia and empyema were found at autopsy (fig. 12). In the slides of the esophagus there were numerous areas of infection and necrosis. There was an exudate over the necrotic area, and bacteria crowded the exudate and the esophageal tissues beneath. The second

case is one of infectious thrombophlebitis (fig. 13). The slide showed three microscopic periesophageal abscesses and an abscess of the esophageal wall (fig. 14). The third of the cases of infection of the blood stream is one of mesenteric thrombosis (figs. 15 and 16). In this case the esophagus was infected, the glands were dilated, and in one place an infected and blocked duct had broken down into an abscess.

Pneumonia.—Of thirteen cases of pneumonia, one central, two lobar and ten bronchopneumonia, six showed acute infection, three

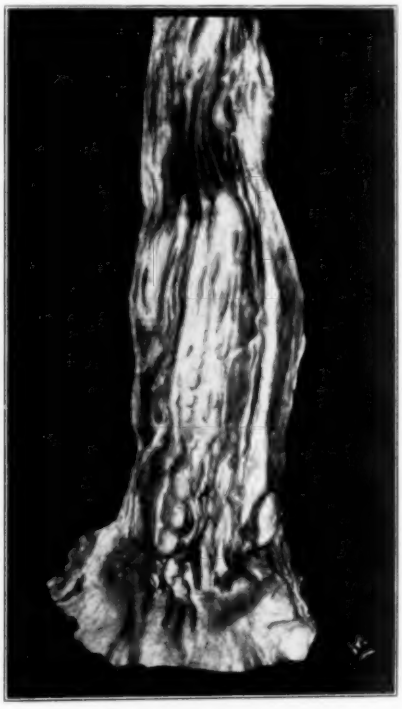


Fig. 13.—The photograph shows a discolored area in the esophagus at the junction of the middle and lower third. At the lower end the epithelium is wanting in patches, and there is again discoloration. The microscopic sections from the lower end of the esophagus show periesophageal abscesses and an abscess in the esophageal wall.

chronic infection and two periesophageal hemorrhage. The gland ducts were enlarged, probably from infection, in two cases.

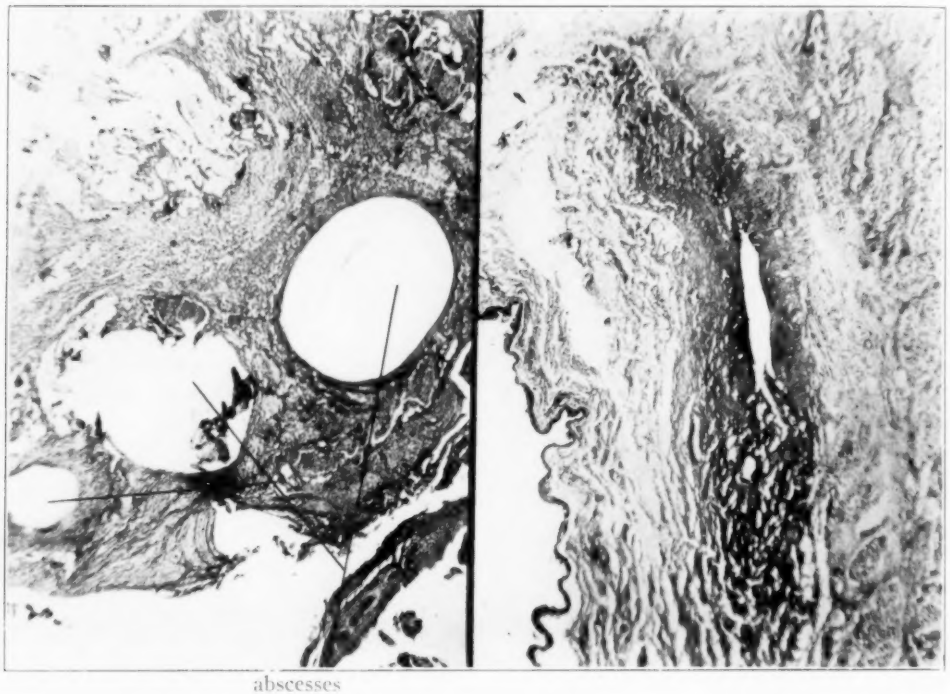
In the case of central pneumonia the microscopic slides showed three superficial veins obliterated with thrombi (fig. 17). The thrombi were made up mostly of small round lymphocytes.

General Peritonitis.—Of three cases of general peritonitis, one showed marked infection of the esophagus (figs. 7 and 18).

Chronic Disease with Acute Infection.—There was one case of duodenal ulcer with hemorrhage. The slide from this case showed a fibrinous exudate under the epithelium equal in width to the combined thickness of the circular and longitudinal muscular layers. The exudate showed acute infection (fig. 1).

The sections in a case of purulent ascending pyelonephritis showed a subepithelial and periglandular infiltration with small lymphocytes, and infection under the epithelium, that is, chronic infection (fig. 19).

In one case there were starvation, dehydration and an acute exacerbation of chronic nephritis. The slides showed acute infection (fig. 20).



abscesses

Fig. 14.—The first photograph shows three periesophageal abscesses. The second photograph shows an abscess in the esophageal wall near the periesophageal abscess; × 23.

In a case of arteriosclerosis, a specimen taken from the lower end of the esophagus showed acute necrosis of the mucosa of the esophagus with polymorphonuclear leukocytes and bacteria in the necrotic area.

In a case of cholecystitis in which operation was performed, there were multiple abscesses of the liver. The general microscopic observations were as follows: There were thickening of the subepithelial connective tissue and a subepithelial infiltration of small round lympho-

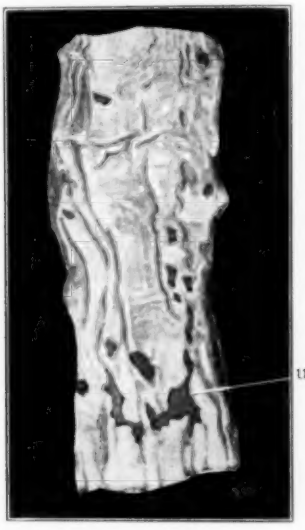


Fig. 15.—Photograph of the lower end of the esophagus, showing sharply defined ulcers. The whole esophagus showed a marked infiltration with lymphocytes. (Photograph by Dr. Ireland.)

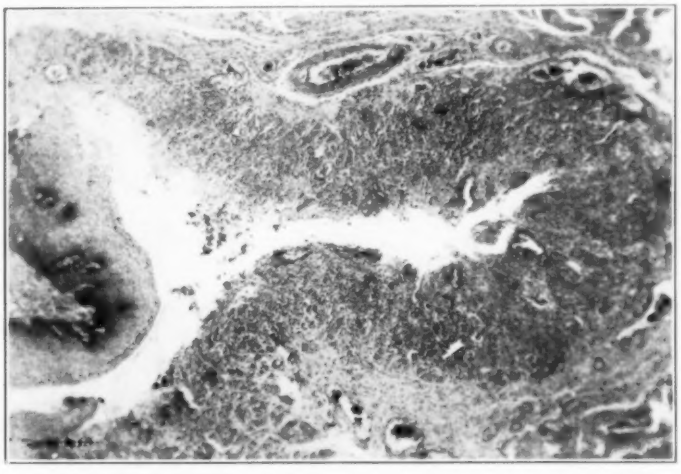


Fig. 16.—This photomicrograph shows a superficial ulcer of the esophagus. Other slides showed chronically infected glands. In one slide there was a blocked and infected duct which had broken down into an abscess.

cytes. In places enough polymorphonuclear leukocytes were present to indicate infection. There was dilatation of the superficial vessels. Myeloma was found in three sections. In this case, therefore, there were chronic infection and acute infection. Multiple myelomas were present (fig. 21).

The slides showed a marked subepithelial infiltration of lymphocytes and greatly dilated vessels. There was a large myeloma in the circular layer. The esophagus was chronically infected (fig. 21).

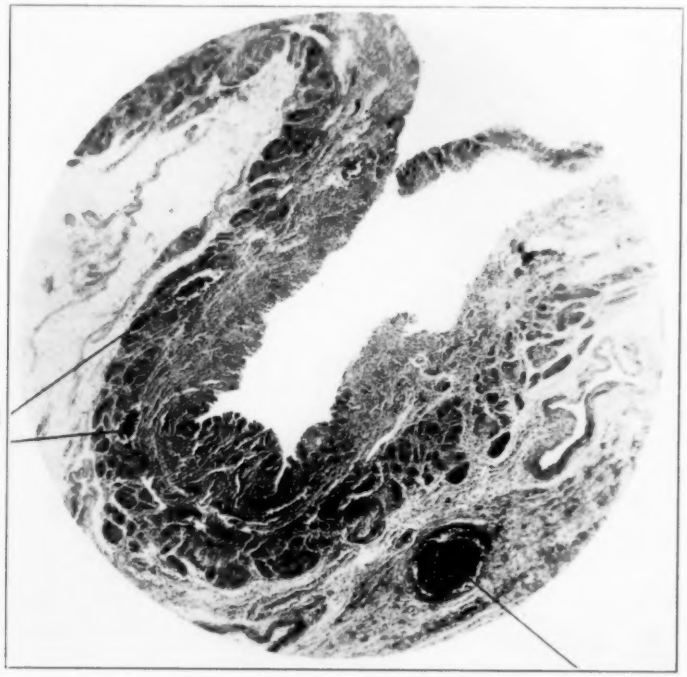


Fig. 17.—The section shows three thrombosed veins.

In a case of general arteriosclerosis the slides showed thickening of the subepithelial connective tissue due to a deposit of fibrin. The gland ducts were dilated. The esophagus showed acute infection and ulceration (figs. 2 and 3).

Cholecystectomy was performed in a case of pyemia in which there were multiple abscesses of the lungs. Unlike the other cases of infection of the blood stream reported in this paper, the esophagus did not show acute but chronic infection. This case is of value, however, because it is another instance of infection of the gallbladder and associated infection of the esophagus.

FLAKING OF EPITHELIUM

In examining the esophagus after death, it is not uncommon to find areas of discoloration. When the epithelium is unbroken, the discoloration is due to enlarged superficial vessels. The same discolorations are even more common in the stomach, and are due to the same cause. Occasionally in the esophagus the superficial vessels are found to be broken and surrounded by hemorrhage. In other words, there may be a discoloration of the esophagus over large areas with the epithelium absent and often somewhat thickened. For years, I considered this

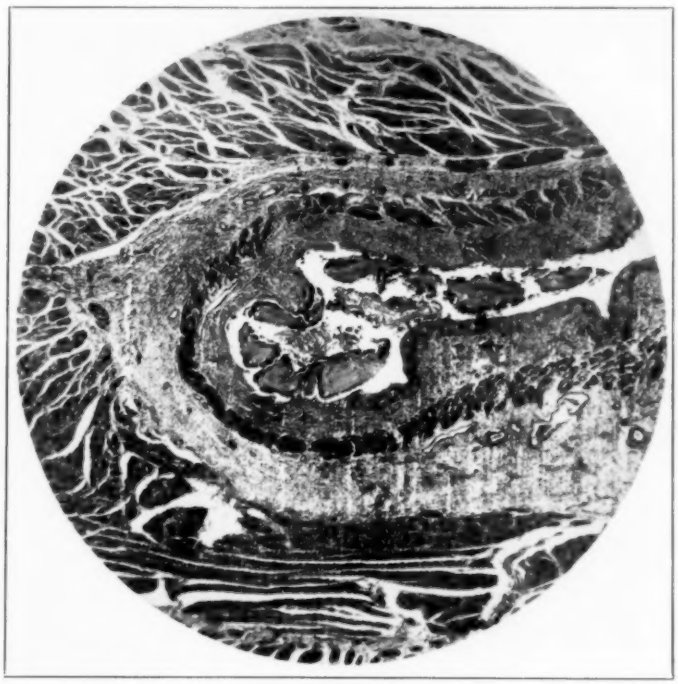


Fig. 18.—The photomicrograph shows an infected superficial ulcer, over which there is a marked exudate.

condition a postmortem change because the mucosa of the esophagus, like that of the stomach, quickly disintegrates. Fortunately, the esophageal epithelium is more resistant than that of the stomach. I was curious to see whether this condition might not be due, in certain cases, to infection. Microscopic examination of sections taken from such areas showed that it is.

The following two cases illustrate this condition:

In the first case, the diagnosis made at autopsy was coronary thrombosis, intramural thrombus and multiple thrombi. The loss of epithelium was associated with chronic infection (fig. 22).

The diagnosis made at autopsy in the second case was that of renal tuberculosis. There was an extensive loss of epithelium. Microscopic examination showed an acute infection (polymorphonuclear leukocytes) associated with a chronic infection. There were an increase of subepithelial connective tissue and a marked massing of lymphocytes in the subepithelial stroma (figs. 23 and 24).

Fig. 19.—The sections showed subepithelial and periglandular infiltration with small lymphocytes, that is, chronic infection. This illustration shows two masses of lymphocytes under the epithelium.

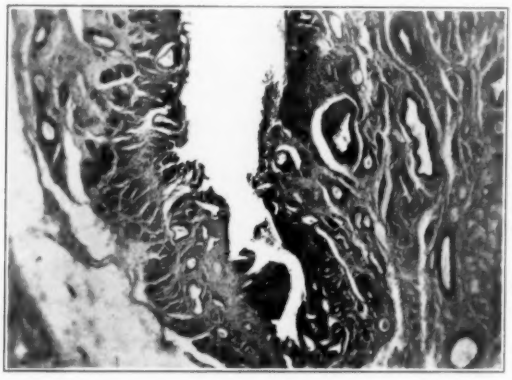


Fig. 20.—The photomicrograph shows acute infection. The epithelium is lost, and the subepithelial tissue is infiltrated with polymorphonuclear leukocytes.

The old names for these cases, I suppose, would be acute and chronic esophagitis.

The majority of the cases in this series were in patients with arteriosclerosis. They were inmates of a state hospital for the mentally defective, and there were many elderly people among them. As one would expect, the majority showed sclerotic changes in the arteries. What interested me more than this, however, was the fact that there

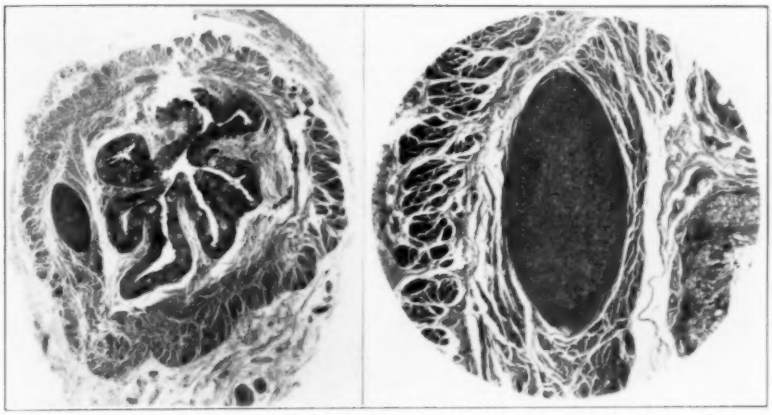


Fig. 21.—The photomicrographs show a great dilatation of the superficial vessels and a pronounced infiltration with lymphocytes. These two findings indicate chronic infection. There is a myoma in the circular layer. The section is taken from the lower end of the esophagus. The first illustration is a low power view, showing the full section of the esophagus. The second photograph is a high power view of the myoma.

Fig. 22.—The photograph shows a loss of epithelium in the lower third of the esophagus. The microscopic slides showed an infiltration of lymphocytes where the epithelium is lacking. The esophagus, therefore, shows chronic infection.

were many instances in this group of chronic infection of the esophagus as shown by subepithelial infiltration with lymphocytes, dilated gland ducts with lymphocytic infiltration about them and infiltrations of lymphocytes into the glands themselves. In addition, I found a number of instances in which there was a moderate infiltration of lymphocytes into Auerbach's plexus. I had not found this before (fig. 25).

Having shown some years ago that the esophagus can be infected from without, to find that it is often infected from within boxes the

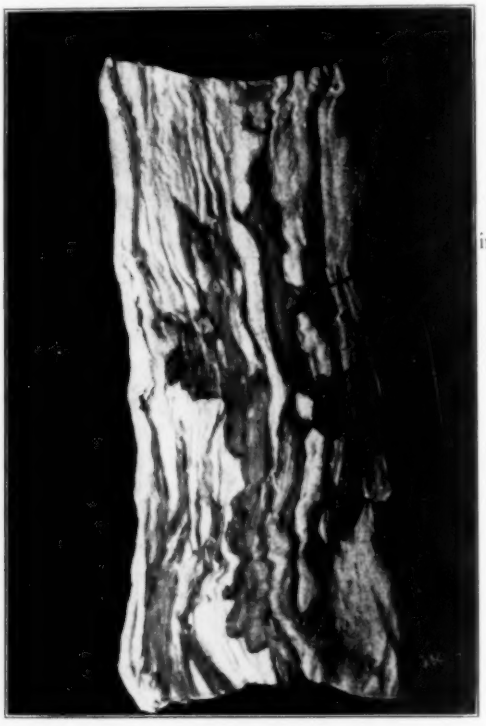


Fig. 23.—Photograph of the lower two thirds of the esophagus, showing extensive loss of epithelium. Microscopically, the esophagus showed an increase of subepithelial connective tissue and a marked infiltration of lymphocytes. Polymorphonuclear leukocytes were abundant. The esophagus is acutely infected. (Photograph by Dr. Ireland.)

compass, so to speak, and brings the story to a natural end. The pathologic changes which are common in other organs of the body are common in the esophagus. No one, however, has taken the trouble to work in this restricted field and to record them.

Cirrhosis of the Liver.—It is old knowledge that the esophageal vessels are greatly dilated in cases of cirrhosis of the liver due to back

pressure. It has also long been known that the dilated vessels at times bleed, even enough to cause death. The rupture, even in fatal cases, may be so small that it cannot be located at autopsy. Figure 26 is from such a case.

The specimen in figure 27, taken from a case of atrophic cirrhosis of the liver, was 10 inches (25.4 cm.) long. The upper 2 inches (5 cm.) were soft. The remainder of the specimen was hard, almost like

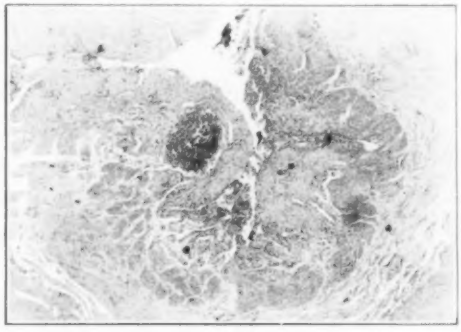


Fig. 24.—This photomicrograph was made of a section through a superficial ulcer. It shows an acutely infected ulcer with exudate.

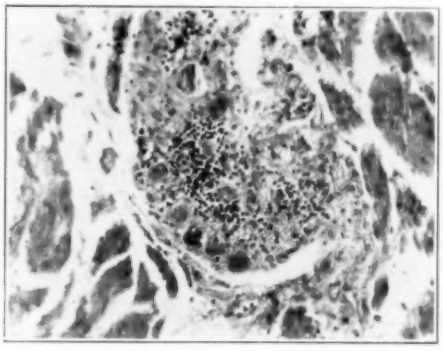


Fig. 25.—The section shows a slight infiltration of Auerbach's plexus with lymphocytes. I have found a similar infiltration in this series of specimens, perhaps five or six times. I have not yet found an infiltration with polymorphonuclear leukocytes. I still think that involvement of Auerbach's plexus in cardiospasm is a secondary and not a primary affair.

cartilage. At the lower end there were a number of enlarged glands. One measured fully 1 cm. in each diameter. On section, the whole of the firm part of the esophagus was seen to be studded with cystic glands (fig. 28).

On microscopic section, the gland ducts were found to be greatly dilated. Occasionally one was found to be filled with pus. Fibrosis of the circular muscular layer occurred in places (fig. 29).

In this case, then, there were dilated and infected gland ducts and fibrosis of the muscular walls of the esophagus. The cystic ducts plus the fibrosis of the muscular layer gave the esophagus its cartilaginous feel.

The gross specimen in figure 30, taken from a case of atrophic cirrhosis of the liver, was 8½ inches (21.59 cm.) long. There were

Fig. 26.—The upper figure shows greatly dilated esophageal veins. The lower figure also shows dilated veins in the stomach. In one place there is a streak of hemorrhage reaching the surface. This patient died of hemorrhage. At autopsy, the bleeding point could not be found. Often it cannot be determined.

many subepithelial hemorrhages. The lower end was much discolored, with large areas bulging into the lumen of the esophagus which, on section, looked like hemorrhage. A few areas looked like ulceration.

The microscopic sections showed greatly dilated veins; they were dilated almost to the bursting point, especially at the cardia. At the cardia there was a gland. A section of the esophagus was taken at this point. The muscles of the esophagus, where the gland was attached,

were thin and mostly replaced by fibrous tissue. There were areas of fibrosis about the enlarged superficial vessels. There were not many enlarged gland ducts (figs. 31, 32 and 33).

Periesophageal Hemorrhage and Hemorrhage Into the Esophagus.—The specimen taken from a case of adynamic colon was 9 inches (22.86 cm.) long. The esophagus was slightly dilated. There were two areas of subepithelial hemorrhage.

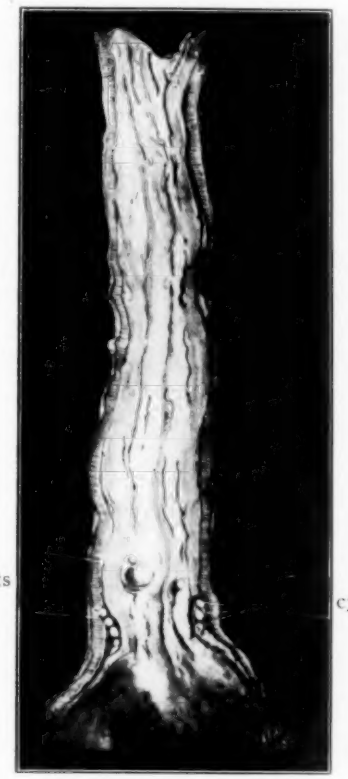


Fig. 27.—The illustration shows cysts projecting into the esophageal lumen and cysts under the mucous membrane. In chronic infection of the esophagus, infected glands and cystic gland ducts are common.

Gross examination of the esophagus showed a hemorrhage at the upper end which extended through both muscular layers. Three inches (7.6 cm.) above the cardia there were subepithelial and periesophageal hemorrhages (figs. 35 and 36).

I have spoken about subepithelial and periesophageal hemorrhages in a previous paper.[1] The finding of more specimens of this condition

1. Mosher, H. P.: Hemorrhage Into the Esophagus at Birth and in the Adult, Laryngoscope **41**:591 (Sept.) 1931.

Fig. 28.—The illustration is a cross-section of the esophagus shown in figure 27. The esophagus is riddled with cysts.

Fig. 29.—The photomicrograph shows two enlarged gland ducts. The larger one is surrounded by a ring of small lymphocytes and contains a large clump of polymorphonuclear leukocytes.

shows that it is not a rare happening but must be reckoned with in the pathologic changes which occur in the esophagus.

In the previous paper I gave as a possible explanation of the hemorrhages that they might be hemorrhages associated with sepsis. I also stated that they were often due to back pressure and were especially common in cirrhosis of the liver. My present belief is that back pressure is the common cause. It is easier to prove that back pressure is a cause than that sepsis is.

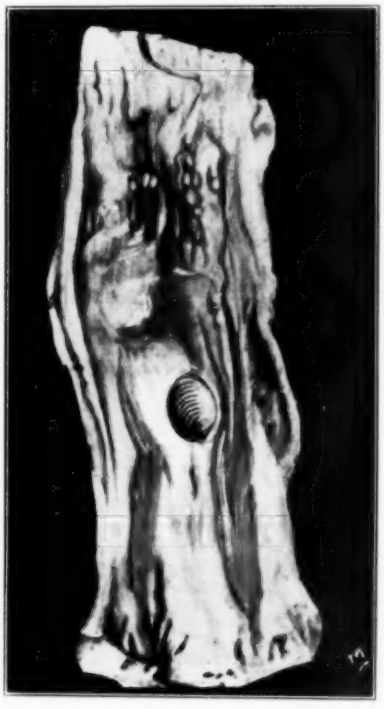


Fig. 30.—The illustration shows a number of small cysts and one large cyst under the epithelium of the esophagus.

Prostatectomy was performed in one case. The patient had bronchopneumonia. Seven inches (17.78 cm.) from the cardia, where the esophagus was thin, an area of ulceration and infection was present. There was also periesophageal hemorrhage.

Myoma of the Esophagus.—In this series of one hundred autopsy specimens, I found three instances of myoma of the esophageal wall. In one of the cases there were three myomas in a series, showing that these tumors can be multiple (fig. 21).

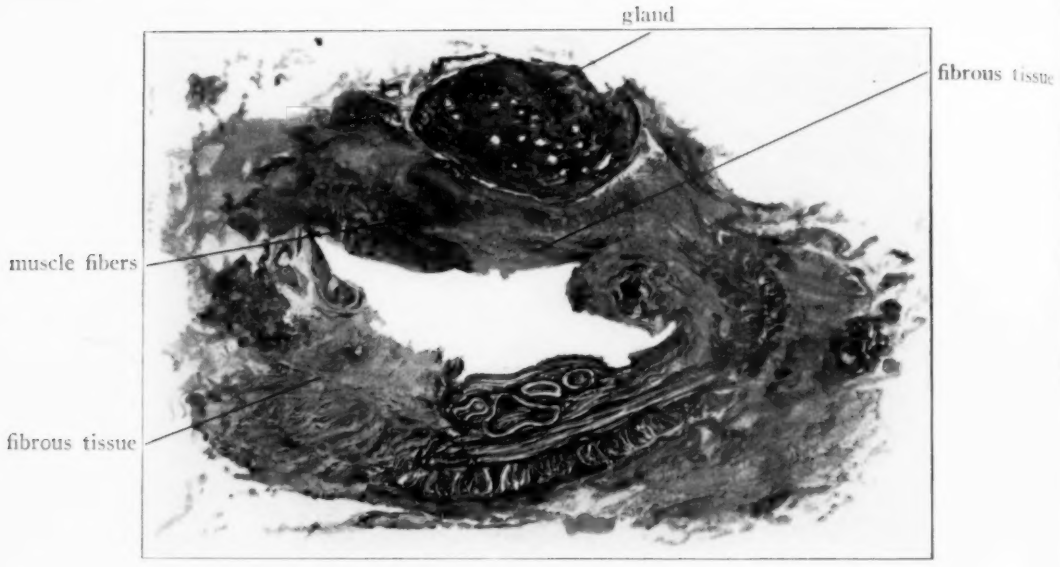


Fig. 31.—The vessels throughout this esophagus were greatly dilated. The slide shows a gland which is adherent to the esophagus. Over this the esophageal muscles are replaced by fibrous tissue. On the opposite esophageal wall, the enlarged superficial vessels are surrounded by dense fibrous tissue.

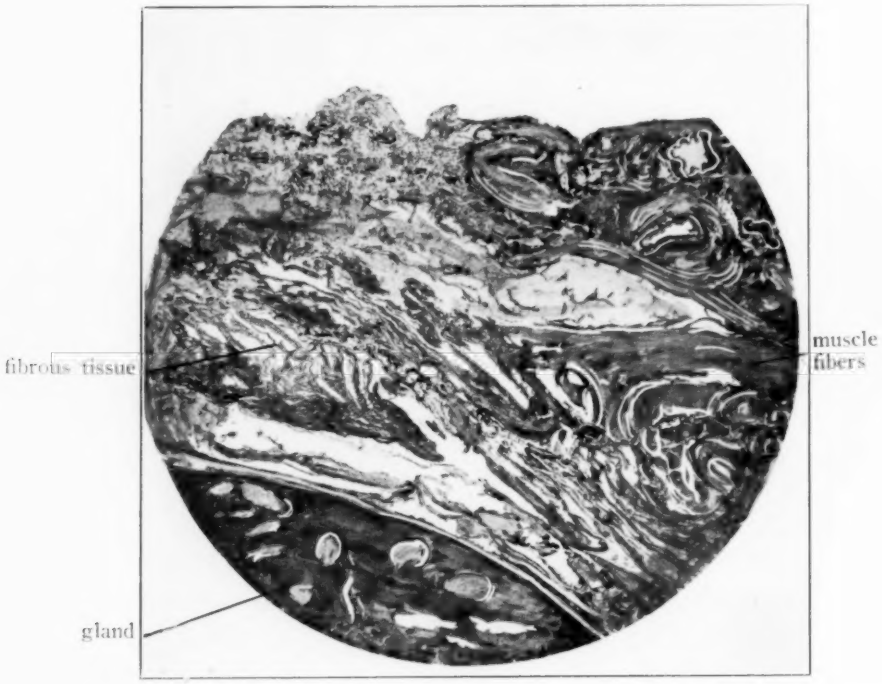


Fig. 32.—The photomicrograph shows the edge of a periesophageal gland. Opposite this the muscle fibers of the esophagus are almost completely replaced by fibrous tissue. The epithelium is lost. In the subepithelial tissue the dilated vessels are surrounded by thick connective tissue.

Previously, and in another set of autopsy specimens, I found my first case of multiple myomas. Such growths, therefore, are not excessively rare. Their importance lies in the fact that they are the same in structure as myoma of the uterus, and presumably have the same possibilities of growth. They become, therefore, another possible cause of esophageal obstruction.

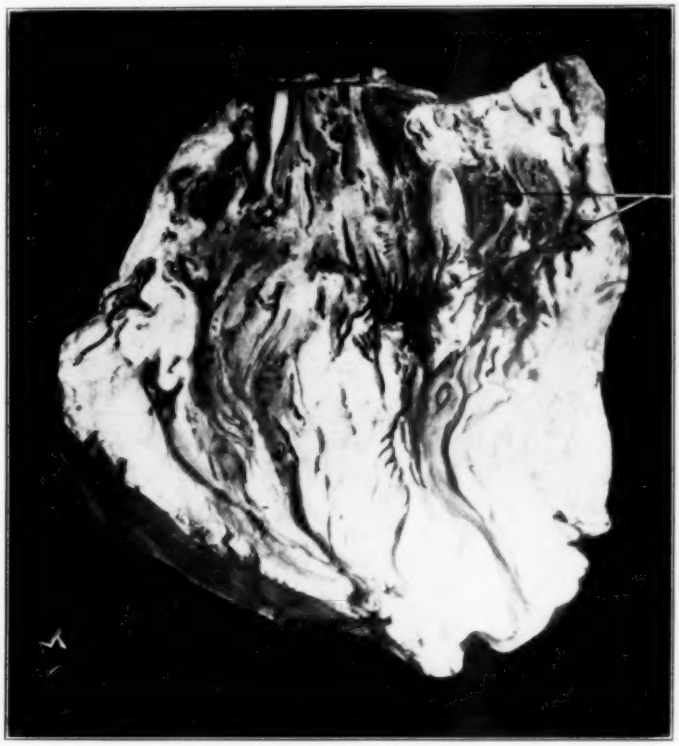


Fig. 33.—The illustration shows the lower end of the esophagus. The dark areas are submucosal hemorrhages. There was a similar area in the upper part of the esophagus. These areas appear to be near the breaking point.

BLOCKING OF THE GLAND DUCTS BY THE DOWNWARD EXCURSION OF THE SURFACE EPITHELIUM

I mentioned two cases of cirrhosis of the liver in order to illustrate the dilatation of the vessels from back pressure. I refer to these cases again for a second reason, to show blocking of the gland ducts.

In these two cases of cirrhosis of the liver, the surface epithelium was seen to have grown down the gland ducts, narrowing their lumen, and in places blocking the duct. This is a new finding in the esophagus.

I reported it a year ago. This peculiar behavior of the epithelium occurs in the breast in Paget's disease (cirrhous carcinoma). Dr. T. B. Mallory, pathologist at the Massachusetts General Hospital, has never seen this condition in the esophagus. In the breast it is considered a precancerous change. I have found it in the esophagus too often to think that it is always to be regarded in this light (figs. 2 and 37).

SOLITARY FOLLICLES AND THE GLAND DUCTS

In the routine examination of sections of the esophagus, I noticed a cross-section of a gland duct squarely in the middle of a solitary follicle. This was thought to be a chance happening, but further search brought to light many other sections which showed the same phenomenon. This finding raised the questions as to how solitary follicles are formed and

Fig. 34.—This figure shows fibrosis of the muscularis mucosa. In chronic infection of the esophagus, disappearance of the muscularis mucosa from fibrosis is quite common.

what their function is. It has long been known that they increase in number in infection, especially chronic infection. It has been supposed that the lymphocytes which form them come from contiguous blood vessels. It is not uncommon to find collections of follicles about blood vessels which are near the surface of the esophagus. It seems from my findings, however, that they can be formed by collections of lymphocytes about gland ducts.

That these ducts are not always seen in the follicles is due to the fact that in cutting the sections, the duct is missed. A fortunate set of serial sections settled this point. Figure 37 shows a superficial gland which is moderately infiltrated with lymphocytes. Nearby is the dilated duct of the gland. There is a collection of lymphocytes about it. This

duct can be followed to the surface of the epithelium. As it pierces the epithelium, there is a collection of lymphocytes on either side of it. The section is cut in the long axis of the duct. If it had been cut horizontally, it would show a section of the duct entirely surrounded by lymphocytes. In other words, if cut horizontally, it would give the picture of a solitary follicle having a section of the duct within it. If, on the other hand, the section had been cut on a slant, the duct would not be visible, and the section of the mass of lymphocytes would appear as the ordinary solitary follicle. This, then, is one method by which an apparent solitary follicle can be formed.

Fig. 35.—The photomicrograph shows hemorrhage into the longitudinal muscular layer. Other slides from this case showed periesophageal hemorrhage.

Up to this point, what I have said has been along academic lines. I should like now to give a few practical points which have come to the surface during the course of a somewhat long, and at times bitter, experience with esophageal cases.

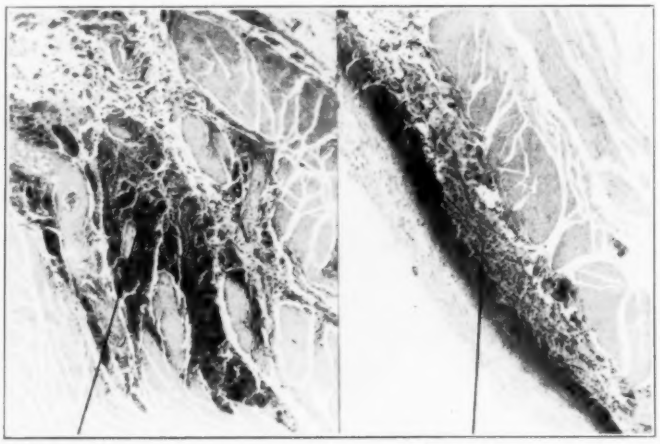
METHOD OF CONDUCTING THE FLUOROSCOPIC EXAMINATION

For fluoroscopic examination it is essential that everything should be in readiness and in working order. This is determined by testing the instruments on the table as they are assembled. The examination is a gaggy and, at times, a messy performance. It must go quickly and systematically if the operator expects the patient to submit to it a second time, that is, if progressive dilatation is to be carried out. This, of course, is the aim of the treatment. Figure 38 shows the set-up of the fluoroscope and the table for instruments.

The examination is naturally divided into two parts: the fluoroscopic examination of the chest and the esophagus and the fluoroscopic passage of the bag, and the determination of the site and extent of the stricture and the ease with which it can be dilated. Before passing the bag, if there is a high fluid level and the esophagus cannot be made to empty itself by the giving of a compound effervescing powder, it is better to empty the esophagus by a stomach tube. Otherwise, fluid will splash over into the larynx and make the examination a distressing affair, if it does not stop the procedure.

FLUOROSCOPIC EXAMINATION OF THE CHEST AND THE ESOPHAGUS

The fluoroscopic examination of the chest is carried out in the familiar routine fashion. First the position of the diaphragm and the



hemorrhage hemorrhage

Fig. 36.—The first figure shows intramuscular hemorrhage; the second, periesophageal hemorrhage.

amount of movement are ascertained. The presence of adhesions in the costophrenic angles or between the diaphragm and the pleura is determined. The size and position of the heart are made out, and it is determined whether the arch of the aorta is enlarged. Thickening is looked for at the hilus and the apexes of the lungs. The patient is given a mouthful of barium sulphate. As this is swallowed, the level of the fluid line is made out. It is usually about 1 inch (2.5 cm.) below the arch of the aorta. The barium is watched from the moment it passes the base of the tongue. It accumulates first at the base of the tongue. The epiglottis can be seen to bend downward and to snap quickly back. If there is no narrowing of the esophagus, the barium quickly reaches the level of the fluid, floats there a second and then

breaks off in chunks and sinks downward. At the lower end of the esophagus, it accumulates in the typical awl-like point if there is fibrosis. If not, it enters the stomach freely, but when the diaphragm descends the esophagus is shut off for a fraction of a second. If the swallowing of the barium is watched with the patient in the anteroposterior position, the stream of barium is seen to divide below the base of the tongue into a right and a left stream, one for each pyriform sinus. These streams quickly join at the bottom of the cricoid cartilage. The movements of the epiglottis and the streams of barium in the pyriform sinuses have to be watched for closely, because they are quickly over. The patient is told to take a deep breath in order to see how the lung triangles light up, that is, to see whether there is thickening, and whether the descent of the diaphragm closes the esophagus as it should. If the

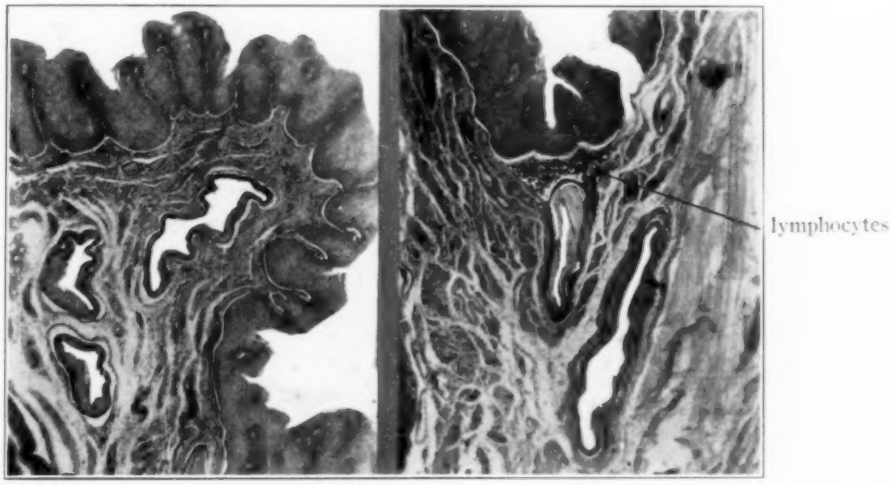


Fig. 37.—The first photograph shows enlarged gland ducts making their way to the surface of the epithelium. The epithelium of the ducts is much hypertrophied. In the second photograph a duct has almost reached the surface. The upper end of this is surrounded by a mass of lymphocytes. If the microscopic section had been made through the collection of lymphocytes and without cutting the duct, the mass of leukocytes would present a typical solitary follicle.

barium does not enter the stomach readily, the descent of the diaphragm appears to amputate half an inch or an inch of the lower end. If the barium halts at the lower end of the esophagus, the fluoroscopic examination is brought to a close by giving the patient a compound effervescing powder. The two objects of the examination are to find out whether there is a pathologic condition of the chest which can influence the caliber of the esophagus and where and how much the esophagus is obstructed. As was pointed out in describing the fluoroscopic exami-

nation, the esophagus is seen to close when the diaphragm descends. This is a normal happening. It took me a long time, in fact years, to determine where the point of physiologic closure occurs. I finally found that it is at the upper border of the left crus, in other words, at the beginning of the crural canal. Between the point of closure and the stomach, there is often a gap or a thin ribbon of barium. This gap represents the width of the crural canal.

USE OF COMPOUND EFFERVESCING POWDER

In the fluoroscopic examination of patients with fibrosis of the terminal portion of the esophagus, the barium halts for half a minute or a number of minutes before it enters the stomach. Meanwhile the

Fig. 38.—The illustration shows the position of the instrument table, the patient and nurse and the vertical fluoroscope.

esophagus ends in the typical awl-like point. In such cases I have the patient swallow a compound effervescing powder. The expansion of the gas in the esophagus will often cause the barium immediately to enter the stomach. The interpretation of this happening is that the pressure of the gas straightens out a certain amount of backward bend or twist of the terminal portion of the esophagus. If the esophagus does not empty after the powder has been given, I conclude that there is a more complete fibrosis than in the ordinary case. It is a great convenience to be able to clean the esophagus of barium before passing the bag. If there is much barium in the esophagus, the surgeon cannot follow the dilatation of the bag.

When there is carcinoma of the lower end of the esophagus or of the fundus of the stomach, the compound effervescing powder is again

of use because the large gas bubble which it makes in the stomach to a certain extent surrounds the growth and causes it to be much more distinct.

The barium striped bag with its flexible finder is passed with the aid of the vertical fluoroscope. The machine is placed on a platform about 2 feet high. The patient stands on the front part of the platform behind the fluoroscopic screen. A narrow movable platform of the same height and the same length is placed on the left of the platform on which the machine is mounted. This second platform acts as a step by which the patient reaches the platform on which the fluoroscope is mounted. It also serves as a platform for the assistant who holds the air gage and inflates the bag to the pressure called for by the operator.

These two platforms place the operator and the assistant on the proper level as regards the patient. They enable the roentgenologist and the operator to see the fluoroscopic screen without stooping, yet the operator can reach the staff of the barium bag easily and can manipulate it at will. The fluoroscope is at the right height for taking plates easily (fig. 38).

When the bag is passed for the first time in a new patient, it may be passed only partially. In other words, the patient may have to be trained. The operator has to get the patient's confidence and keep his own. Exceedingly nervous patients or patients who cannot understand what is said to them had better be examined under ether.

The patient sits in a chair which is placed between the instrument table and the fluoroscope (fig. 39).

In passing the bag, it is better to let the patient swallow the finder himself. If the surgeon attempts to guide it by putting his finger in the pharynx as he does with an elastic bougie, he is liable to push the end of the finder into the larynx. I found this out when I first used the bag. When the patient has swallowed the finder beyond the cricoid cartilage, the surgeon pushes the bag down until it also is beyond the cartilage. The bag always sticks a little until it passes this point. Then the patient mounts the platform and stands behind the fluoroscopic screen. The assistant steps on the second platform and attaches the air bag and the manometer to the tubing running to the bag. The lights are then put out, and further passage of the bag and finder is accomplished under the fluoroscopic control. If the finder curls on itself, the bag is withdrawn sufficiently to allow it to straighten out, and then the bag is again carried down. It is easy to see the position of the finder at all times.

If the esophagus is not much dilated and does not bend sharply to the right, the finder usually enters the stomach readily. Once it is in the stomach, the bag can be safely carried down. When

about half of the bag is in the stomach and the other half is in the lower part of the esophagus, the assistant is told to inflate the bag with 1 pound of pressure. If the barium lines on the bag are fresh, the operator easily sees the site of the stricture and its vertical extent. At this point two plates are usually taken, one in the lateral position and one in the antero-posterior position; or two plates are taken in each position, one with the patient holding his breath after exhaling and the other after inhaling.

Fig. 39.—The illustration shows the grouping of patient, nurse and operator for passing the diagnostic bag.

If the patient complains of pain, even at as low pressure as one-half pound, the dilatation is stopped (fig. 40).

This is generally enough for the first examination, for both patient and operator, because the entire procedure is done under tension and must be performed quickly. The team-work by the nurse, assistant, operator and roentgenologist must run smoothly.

The developed plates give the exact location of the fibrotic narrowing and show the amount of dilatation obtained. At the second dilatation,

which usually is carried out about a week later, the pressure is raised to 1½, 2 or even 3 pounds, according to the lack of pain indicated by the patient, the ease with which the esophagus dilates and the extent to which it dilates. Dilatation to 1 inch, which is the normal width, is considered satisfactory. In the light of the autopsy specimens that I have accumulated, some of them my own tragedies and other specimens sent to me, some of them also tragedies, but by no means all, I am no longer content to try for quick relief by overdilatation, as I have found that three or four dilatations during a period of six weeks or two months will give a clinical cure in most cases, and that subsequent dilatations are called for only at periods of three months, six months or even a year.

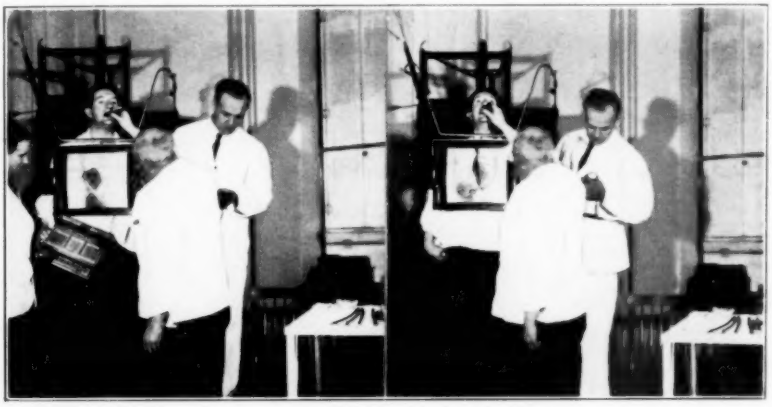


Fig. 40.—In the first figure the roentgenologist is about to take a plate. In the second figure the diagnostic bag is in place, the assistant is inflating it, and the operator is watching the dilatation on the fluoroscopic screen.

In the past I have employed overdilatation, but since I have been using the barium striped bag, I have not found it necessary. I no longer use the mechanical dilator which is called by my name. This I say with apologies to Mr. Mueller. Years ago I gave up using the hydrostatic dilator. Ugly rumors still hover about it.

When the patient is too nervous to allow the passage of the bag, or when he is in poor condition but not desperate and needing an immediate gastrostomy, I examine him under ether anesthesia with the esophagoscope and dilate the stricture with bougies. I am content with a slight dilatation. A bougie is like a bullet, and one cannot tell where its far end is. The examination under ether gives me the lay of the land. Of course, if there is a suggestion of ulcer or of malignant disease, the patient is examined at once with the esophagoscope. If the examination under ether rules out these two conditions and shows the central narrowing typical of fibrosis, I pass the bag about a week later.

The flexible finder makes the bag safe to pass, and its heavy end makes it possible to enter the stomach even in cases in which the esophagus is greatly dilated and has a double bend at the lower end, that is, if the operator has patience enough and will try a second time if he fails the first. There is no reason why a string cannot be used as a guide. So far, however, I have not found it necessary to resort to this method.

I have been greatly surprised that in the majority of cases of fibrosis of the terminal portion of the esophagus, the spiral wire finder, the tip of which is fully 1 cm. in diameter, will pass to the stomach. This means that the barium picture is misleading. From this it appears that the esophagus, ending as it does in all of these cases in the typical awl-like point, is actually closed. Fortunately, as the finder proves, it is not. Its apparent closure is due to a backward bend in the terminal portion of the esophagus which occurs in all long-standing cases of fibrosis of any duration. Recognition of this fact has been a milestone in my dealing with these cases.

In my experience with cases of fibrosis of the terminal portion of the esophagus, the finder passes to the stomach except in some of the long-standing cases. In these cases, when the esophagus is much dilated and sags to the right, and the lower 3 or 4 inches (7.6 or 10.16 cm.) turn sharply to the left and lie flat on a motionless diaphragm, and when the terminal portion turns back to the right, in other words, when there are two bends in opposite directions, it is not only difficult but dangerous to pass the bag.

One cannot tell how much one can dilate the crucial fibrosed area of the esophagus, that is, the crural portion, until one tries. I have been surprised how readily and with how little pressure the area dilates in most cases. I seldom use more than 3 pounds of pressure. This pressure, when applied three or four times, will usually dilate the esophageal lumen to its normal diameter, namely, 1 inch. I do not carry the dilation beyond this point. I have specimens to show that the esophagus in such cases is very thin and is scarred by old ulcers. I believe that in the future such cases will be handled by an anastomosis between the lower end of the esophagus and the fundus of the stomach. If an attempt to pass the bag is decided on, a swallowed string to guide the finder is safer. Woe to the patient if any one attempts to pass a bougie blindly. Dead man's curve, that is, the point where the esophagus turns sharply from right to left at the level of the diaphragm, is sure to justify its name during some such attempts.

The great difficulty in making an anastomosis between the lower end of the esophagus and the fundus of the stomach is the deep and narrow field in which the surgeon has to work. In a case in which Dr. Churchill of Boston operated for me, he found that by putting the

left half of the diaphragm at rest by phrenicectomy and by turning the left lobe of the liver upward and to the right, a surprising exposure could be obtained. It has long been known that the lower 3 or 4 inches of the esophagus can be mobilized and drawn into the abdomen. That the left lobe of the liver can be turned upward out of the way without fracturing it is new knowledge. This procedure gives a comfortable operating field. Its adoption means a great advance in surgical intervention at the lower end of the esophagus.

FINDINGS WITH THE BARIUM BAG

In cases of fibrosis of the terminal portion of the esophagus, the constant finding is a narrowing at the top of the crural canal or a narrowing of the whole canal. This narrowing is due to fibrosis in the esophagus or to fibrosis of the periesophageal connective tissue. When the diaphragm is motionless and in a ptotic position, the narrowing or stricture may be above the level of the diaphragm. I believe that in these cases the stricture originally was in the typical place, at the beginning of the crural canal, but with falling of the diaphragm it has, as it were, become skied.

To repeat, the typical finding in fibrosis of the terminal portion of the esophagus is a fibrotic narrowing at the beginning of the crural canal. This is not shown by the examination with barium, but is clearly brought out by the dilated barium striped bag.

THE BOUGIE WITH THE FLEXIBLE FINDER

The flexible finder attached to the barium bag has made it possible to pass the bag safely and by sight. It is always easy to see the end of the finder, and it is only when the tip of the finder has been maneuvered into the stomach that the bag is advanced. After the examination of patients with fibrosis of the terminal portion of the esophagus under ether, if one wishes to pass a bougie instead of the bag, a bougie with a flexible finder is used. This allows the bougie to be passed by sight. Bougies of varying sizes up to 40 are fitted with a tip which takes either the short or the long flexible finder. These bougies have been a great comfort to me, because, as I have said, they can be passed by sight. They can be used in dilating fibrous strictures of the esophagus which occur anywhere in the lower half. The operator first ascertains by the examination under ether what size bougie the stricture will take and then without ether uses the various sizes with a flexible finder.

SUMMARY

In addition to the old finding that fibrosis of the esophagus, especially of the terminal portion, is the result of infection from contiguous

organs, it has been shown that fibrosis of isolated areas is fairly common, especially in such chronic infections as arteriosclerosis. In infections of the blood stream, the esophagus is often involved to the extent of ulceration. In acute infections (pneumonia) the esophagus may be infected. Chronic infection, as shown by an infiltration of lymphocytes under the epithelium and in the glands and about the gland ducts, is also common. Dilatation of the subepithelial blood vessels is almost constant in diseases such as cirrhosis of the liver which impede the venous circulation. I have stated in previous papers that cirrhosis of the liver and infection of the gallbladder are among the chief causes of infection of the esophagus. As autopsy specimens of these conditions accumulate, this fact is becoming more and more evident. Hemorrhage into the muscular layers extensive enough to disrupt them may occur when there is back pressure on the esophageal vessels. The glands of the esophagus are especially liable to infection, and are probably the chief route by which the esophagus is infected from within. One form of solitary follicle is simply a collection of lymphocytes following a gland duct to the surface. The esophagus, to recapitulate, can be infected from within and from without, and is often infected in both acute and chronic diseases. In the esophagus, fibrosis follows infection, as it does in other organs of the body. As the esophagus is so frequently infected, there is abundant cause for strictures or fibrosis to occur in any part.

Dr. William Freeman of the Worcester State Hospital supplied practically all of the esophageal specimens which furnish the basis for this paper. Dr. William E. Murphy helped with the specimens. Dr. T. B. Mallory and Dr. Percy E. Ireland checked the pathologic findings. Dr. Werner Mueller and Dr. F. Pollock supplied the photomicrographs.

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FUNDAMENTAL PRINCIPLES OF FUNCTIONAL HEARING TESTS

WITH RECENT DEVELOPMENTS IN TUNING FORKS AND SOUNDING RODS

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To use an old phrase, it would certainly be carrying coals to Newcastle to recite in detail before this group the various tests employed in examining the deaf patient. However, a discussion of the newer developments in tuning forks and sounding rods may not be amiss when appended to a brief review of the fundamental facts and methods usually employed. The purpose of these tests of hearing is to determine the presence of impaired hearing and the degree thereof, and second, to decide on the location of the hearing defect, that is, whether it is in the perception or in the conduction apparatus. For the purpose of such testing, a number of methods and appliances are used, such as the voice, acoumeter, watch, Koenig rods, tuning forks, resonators, whistles, monochords, audiometers and sounding rods. The observation of the patient is important, for much may be learned regarding the state of the person's hearing even before the tests are begun. For instance, the loudness of the voice may be used in differentiating between the impairment of conduction and perception. When there is decided middle ear deafness, for instance, otosclerosis or chronic tubal involvement, the patient has a marked autophonia; his own voice seems very loud to him and, believing that it likewise sounds loud to others, he tends to speak very low. On the other hand, when there is a pronounced change in the perception apparatus, the bone conduction is materially diminished; his own voice sounds very feeble to him, and in order to make others understand, he often talks very loud. There is a tendency to turn the better hearing ear toward the speaker, and often the time-honored method of placing the hand in contact with the auricle is employed in order better to understand the speaker's voice. One also notices that patients with impaired hearing watch the lips of the speaker closely, either consciously or unconsciously doing a great deal of lip reading.

Read before the Section on Laryngology, Otology and Rhinology at the Eighty-Fourth Annual Session of the American Medical Association, Milwaukee, June 14, 1933.

Next follows a careful examination of the ear, including the auricle, mastoid region, external auditory canal and drum membrane. Likewise, examination of the nose, nasopharynx and pharynx should be carried out, and the presence of any abnormalities noted which may have influence on the ear, especially by way of the eustachian tube. I believe that my opinion agrees with that of others, that too much emphasis should not be placed on the presence of septal deviation and other forms of nasal obstructions as the cause of involvement of the ear; nor should these alone serve as indications for intranasal surgical intervention unless an obstruction to breathing or a decided interference with drainage of the nasal accessory sinuses calls for relief per se. If it is shown that the inner ear is involved, operation will surely not aid. Only when there is direct interference of aeration of the eustachian tube by very large tonsils, conditions in the nasopharynx, such as tumors, large adenoids or hypertrophied posterior ends of the inferior turbinates, or when pus from the sinuses infects the tube do I believe that one may expect amelioration of the process in the middle ear by operation on the nose, pharynx or nasopharynx.

The voice in the form of an unaccentuated whisper or unaccentuated conversation is one of the most commonly used tests. This cannot be standardized as one would like to have it, but nevertheless from a practical standpoint it is perhaps the most important test, since the criterion of efficient hearing is the ability to understand speech. Every effort should be used to employ the same intensity and pitch, and this is best accomplished by using the residual air. It is important that the subject be tested with his eyes closed or averted from the speaker, that the opposite ear be well occluded with a moistened finger and that no part of the body comes in contact with a wall or other fixed structures, except the floor; otherwise the sound may be carried by way of the bones of the arm, so that hearing will not be entirely by way of air conduction.

It is important, when using the voice, the watch, the acoumeter or some other method whereby one determines the hearing, to approach the patient from afar to the point at which he first begins to hear, and not to produce the sound close to the ear and then move away from him, because it is a psychologic fact that as it recedes from the person, he will from memory often state that he still hears the sound when he no longer does.

It is difficult to occlude the opposite ear completely, but every effort should be made to see that the patient hears the sound only in the ear that is being tested. In order to overcome this difficulty, a 2 or 3 meter speaking tube may be used, the tip of one end being inserted in the patient's ear, the examiner whispering through the other funnel-shaped

end. The sound is then carried only through the lumen of the tube to the examined ear, and if repeated one is certain that the hearing has been by way of the ear that is being tested. After testing with the voice, watch or acoumeter, the ears may be inflated with the Politzer bag, or preferably by way of the eustachian catheter, and the degree of hearing is again determined. The presence or absence of improvement in hearing following careful inflation is important in the determination of the prognosis. If a decided improvement is observed, it usually means that further treatment will benefit the patient; on the other hand, when no improvement whatever is noted, especially after inflation has been repeated two or three times on different days, experience leads one to believe that in most cases local treatment will be of very little avail.

TUNING FORK TESTS

The principal tuning fork tests are the Weber, Schwabach and Rinne.

The Weber test is used for determining the lateralization of sound, and in the normal instance, the sound is heard either in both ears or "in the head," as the patient usually states. The test is made by placing the vibrating fork in the midline of the vertex. In pathologic cases with impairment of the conduction apparatus the sound is lateralized to the worst ear, and if both ears are so involved, it goes to the worse of the two. If there is involvement of the perception apparatus, or if there is a lesion of both inner ears, the hearing of the sound is lateralized in the better ear of the two. Since the test gives unreliable results in many cases, it is of value only when it agrees with and confirms the clinical findings.

The Schwabach test is for the determination of the bone conduction of a subject as compared with a normal standard. Instead of comparing the duration with one's own bone conduction, it is better to determine the average hearing for the particular fork employed with a number of normal subjects and then use that as a standard. In making this test, a vibrating fork is placed in the middle line of the vertex, or some place it at the root of the nose, in order to see how long the fork is heard as compared with the normal standard. The question of the fatigue symptom must always be considered, and therefore the fork must occasionally be raised from the skull, to see whether or not the patient still hears. The duration of bone conduction even in normal persons varies considerably, depending on the age (at or after middle age it is usually considerably diminished, although it may differ in persons of the same age). The thickness of the cranial bones, the size of the air spaces of the mastoid cells and certain anomalies of the skull, such as traumatisms and adhesions between the dura and the bone,

influence the Schwabach test by giving a greatly shortened bone conduction, in spite of the normal hearing by air conduction. Likewise, the amount of hair, the tension of the skin, the contact with the vertex and the force with which the fork is applied make variations in bone conduction. It is important to remember that a slight increase or a slight diminution in the duration of bone conduction does not have any significance.

Politzer stated that from a prognostic point of view, cases with lengthened bone conduction usually have a more favorable course as the result of treatment than those in which it is shortened. But one must not forget that typical otosclerosis, which usually shows prolonged bone conduction, has a very poor prognosis. On general principles, one may say, bearing in mind the various factors that have been previously mentioned, that lengthened bone conduction usually means impairment of the conduction apparatus and a diminished bone conduction usually spells involvement of the inner ear or the auditory nerve.

Oscar Beck of Vienna called attention to the diminution in bone conduction at times noted in syphilitic patients with good hearing and no aural symptoms. He says that this lowered bone conduction may be found in 80 per cent of all syphilitic subjects, and that it appears mostly in the second and only rarely in the first stages. I found this strikingly illustrated in a number of cases of rather early syphilis in which the hearing was apparently still good but bone conduction was markedly diminished and the Wassermann reaction was strongly positive.

The Rinne test was first devised by Rinne [1] in 1855, and is employed for comparing air conduction with bone conduction in the same subject. His method was to place the fork on the upper incisors, and when the sound was no longer heard by bone, the hearing by air was determined. As it is now performed, the test is usually made by placing the vibrating fork on the mastoid bone in the region of the antrum, and when the sound is no longer heard, the prong is held near the external auditory meatus and it is observed how much longer the sound is perceived by air conduction. In the normal person the air conduction is considerably longer than the bone conduction; that is, the Rinne test is positive. In the cases in which bone conduction is longer than air conduction, the Rinne test is designated as negative. Since different forks are used by various otologists, one must remember that the lower the pitch of the fork employed, the more likely is the Rinne test to be negative, and vice versa. There are at least seven or eight varieties of Rinne reactions.

1. Rinne, A.: Präg. Vrtljschr. **1**:72, 1855.

It is therefore important to remember that the mere fact that the Rinne test is positive—namely, air conduction is longer than bone conduction or is negative, or bone conduction is longer than air conduction—does not necessarily show the exact pathologic condition present. The Rinne test is probably the most important of all of these tests, and if one carefully studies the type of positive Rinne reaction which is present, a diagnosis with reference to the location of the lesion, namely whether it is in the conduction apparatus or the perception apparatus or in both, can usually be made.

The varieties of Rinne reactions are:

Two forms of the positive Rinne reaction: (1) Air conduction greatly exceeds bone conduction, giving the normal positive Rinne reaction. (2) Both air and bone conduction are shortened, as in disease of the inner ear.

Three forms of the negative Rinne reaction: (1) Bone conduction is much increased, and air conduction is moderately diminished, as in ordinary disease of the middle ear. (2) Bone conduction is longer than air conduction, but both are shorter than normal; this is often noted in combined disease of the internal and middle ears. (3) Both bone and air conduction are greatly diminished. This occurs in far advanced diseases of the ear because air conduction is then lost faster than bone conduction. (4) The "infinitely" negative reaction, or *Rinne imaginaire*, is sometimes found with one deaf labyrinth, and sound is really heard in the good ear via bone conduction. There is no hearing via air conduction, and sound is heard for only a few seconds via bone conduction. Close first one and then the other external meatus to see whether the sound is heard louder by bone conduction.

Two forms of so-called "indifferent" Rinne reaction (plus-minus Rinne reaction): (1) Air and bone conduction are of equal length because air conduction is slightly diminished and bone conduction is slightly increased. This usually means that a slight involvement of the middle ear is present, and the hearing is quite good. (2) Air and bone conduction are of equal duration, but both are much shorter than normal; disease of the inner ear is usually present, and the hearing is very poor.

Infinite Rinne reaction: If sound is not heard at all by air conduction, but is heard for a short time by bone conduction an "infinitely" negative Rinne reaction is obtained. This is often found in far advanced disease of the inner ear. In two cases I found an infinitely positive Rinne reaction, which is difficult to explain.

The Gellé test is used for determining the mobility of the foot plate of the stapes. Use a Politzer bag or another bag connected by way of tubing and an olive-shaped tip with the external auditory meatus. It is

essential that the ear-piece be so inserted that it is air-tight. With the vibrating tuning fork resting on the bulb, vertex or mastoid, the Politzer bulb or the other bulb is compressed. Normally, there is a positive Gellé reaction, in that the hearing of the fork is diminished while the air is compressed in the external auditory meatus. In this manner the foot plate of the stapes is pushed further into the oval window, and the hearing is impaired for the time being. When the Gellé test is negative, which is the abnormal reaction, the hearing is not diminished by the compression of air because the foot plate of the stapes is fixed and cannot be moved further toward the labyrinth.

The fallacies of this test are: (1) The patient often does not thoroughly understand what is required of him, or (2) he does not pay close attention to the fact that the hearing is somewhat diminished during the time that the bulb is compressed, and (3) it often happens that the ear-piece does not fit tightly into the meatus so that there is an escape of air, and it is then impossible to state whether the foot plate is movable or not. If, however, there is a definite change in the hearing with compression of the air column, it may give valuable information. This is especially true in cases of typical otosclerosis with marked fixation of the foot plate of the stapes.

The Stenger test is used for determining simulation of total unilateral deafness. It is well known that when the two ears are simultaneously exposed to two sounds of the same pitch and intensity, the one that is produced closer to one ear drowns out, or masks, so to speak, the sound that is entering the other ear; that is, if a sound is produced at a certain distance from the right ear, the subject cannot know whether there is a similar sound occurring at a greater distance from the left ear (and therefore apparently of less intensity) than from the right ear. It is essential that a pair of forks of exactly the same pitch be employed, and it is also best that the patient be blindfolded, so that he does not know that two forks are being used at the same time.

I usually employ (following Stenger's suggestion) a pair of Bezold's $a1$ forks (435 double vibrations). Having struck both forks, one of them is held a certain distance from the left ear, for instance, 10 inches (25.4 cm.). In the normal subject, if a fork of the same pitch is placed less than 10 inches from the opposite right ear, for instance, 5 inches (12.7 cm.), he will hear only in the ear from which the fork is a shorter distance, namely, the right one, because the sound coming from that short distance drowns out sound entering the other ear at a distance of 10 inches. If, for example, a patient pretends that his right ear is deaf, test the left or well ear and find the distance at which the fork will be heard, possibly 10 inches. With the right ear, the subject will, of course, deny hearing all sounds, no matter how close the fork is approached to that ear. He hears the sound, but denies having any

perception of it. If one fork is kept vibrating, possibly 4 inches (10.16 cm.) from the right ear, and the other fork approaches to within 8 inches (20.32 cm.) of the left ear, the patient will say that he does not hear any sound at all, even though he previously admitted hearing the fork with the well ear (that is, the left ear) at a distance of 10 inches. The reason is this: The fork, if held in the right ear, will drown out the sound of the fork held 8 inches from the left ear. In view of the fact that the subject claims to be deaf in the right ear, he will say that he hears nothing at all, whereas if he really were deaf in the right ear, he would not hear the tuning fork held near that ear, but would certainly hear the fork vibrating in the vicinity of the left or well ear. Dr. W. A. Wells of Washington some years ago described a modification of this test which is very simple and ingenious. He uses a piece of rubber tubing of ¼ inch (0.64 cm.) caliber, 30 inches (76.2 cm.) in length and of a firmness to give a good conduction of sound. In one end of the tube is an ear-piece which fits well in the auditory meatus, while into the other end is inserted the stem of a long sounding fork of low pitch (about 210 double vibrations per second). After blindfolding the patient and placing the ear-piece in the supposedly deaf ear, one conducts the vibrating fork carefully to the meatus of the opposite ear, not too close, but to a point at which it has been previously ascertained that the patient can distinctly hear it; the really deaf person now reports hearing the fork as soon as it comes into the range of hearing of the good ear. The malingerer, unconscious of any hearing in his good ear because of the lateralization of the sound to the alleged deaf ear (the side of the greater intensity), continues to report no hearing, and this gives incontestable proof of wilful simulation.

With tuning forks as previously employed, one could get a qualitative estimation of the state of hearing, but not a good quantitative one. With the Edelmann-Galton or Schäfer-Galton whistle, and especially with the monochord, the high tones are qualitatively tested. By comparison with a normal standard, an objective measure is obtained as mentioned in the discussion of the Schwabach test. However, for the accurate determination of the degree of percentages of loss of hearing, figured in sensation units, more exact methods are necessary, such as properly calibrated audiometers. A considerable number of audiometers has been perfected. Among these is the one devised by Dr. John Guttman of New York, and another by Dr. Dean and Dr. Bunch, formerly of Iowa City. There is one perfected by Dr. F. W. Kranz, at that time at the Riverbank Laboratory in Geneva, Ill., and another by Dr. Jones and Dr. Knudsen of Los Angeles. The best known and most widely advertised audiometer is made by the Western Electric Company. The latter type has a number of models. The 1*A* is a large instrument for research purposes, with tones from 16 to about 18,000

double vibrations. The *2A* type, which is usually employed for examination in an office, ranges from 64 to 8,192 double vibrations. What is called the *4A* model with a phonograph is used for testing school children in groups. Some of the audiometers mentioned have a continuous tone series, such as the Bunch, Kranz and Jones and Knudsen. The models by the Western Electric Company have tone intervals of one octave. Many otologists use the *2A* model of the Western Electric Company, and find it satisfactory. The audiometer is a valuable instrument, and with it graphs are obtained showing the state of the patient's hearing. The instruments of the Western Electric Company are calibrated on a basis of 120 sensation units for the whole range of hearing. To get the percentage of loss of hearing, the number of units lost is multiplied by eight-tenths. In some instances, however, the curves do not give an absolutely accurate diagnosis. The appliances are heavy, and their cost is high. The *2A* type does not cover the full range of hearing. Audiometers, however, mark an important advance in the instrumentarium of otologists.

It seems to me that great credit is due Harvey Fletcher[2] of the laboratories of the Bell Telephone Company, who has developed the use of the "constant" of damping or decrement of tuning forks for estimating the loss of hearing in sensation units. This constant of damping or decrement is determined by the Bureau of Standards at Washington at a cost of $2 per fork. It is thus possible to use forks and obtain a curve the same as with an audiometer. If a good many forks are used in the testing at various pitches, this method will consume considerably more time than with the audiometer. On the other hand, if a moderate set of forks is used for this purpose, it is much less expensive and is more easily manipulated and transported than is the audiometer. This is not intended, however, to convey the idea that the good qualities of the audiometer are not appreciated. When using the "constant," the fork is given a standard blow and is held near the ear in the usual manner; when the patient signals that he no longer hears the sound, the stop watch is started. The tuning fork is then placed at the examiner's ear, and he stops the watch when he no longer hears the sound. The reading of the stop watch represents the difference in the hearing between the patient and the examiner, and this figure, multiplied by the constant of the fork, gives the loss of hearing in sensation units. In order to make this test more objective, I believe that it is better to find the average hearing of normal persons by air conduction for the particular fork or forks employed, and then simply to note the time that the patient heard the fork and multiply the difference between this hearing and the normal average.

2. Fletcher, Harvey: Some New Methods and Apparatus for Testing the Acuity of Hearing, Laryngoscope **35**:501 (July) 1925.

Among the appliances used for the functional testing of hearing, tuning forks have long played an important part. In order to be efficient, certain characteristics are necessary. Good forks should have the following qualities:

1. They should be made of one piece of metal.

2. The handle should be long enough so that it can easily and yet loosely be held without damping or stopping the vibrations.

3. The forks should sound for a long time.

4. There should be weights on the lower pitched forks to avoid overtones.

5. Forks should not be nickeled.

6. If rustless metal is feasible, it should be used to prevent changes by oxidation.

The best forks have usually been unnickeled, but unless great care is used in handling them and in protecting them from perspiration or the moisture in the atmosphere, rusting takes place and this changes the pitch. On the other hand, when forks are nickeled, the plating sooner or later begins to peel, and the vibrations of the loose pieces of metal give rise to adventitious sounds. For this reason, the Committee on the Standardization of Tuning Fork and Hearing Tests of the American Academy of Ophthalmology and Otolaryngology (of which I have had the honor of being chairman since it was formed in 1921) has been endeavoring, among other things, to find a metal or alloy which is rust proof and at the same time has sufficient elasticity so that it can be used in the making of first-class forks. A number of metals and alloys was investigated, but none of them seemed to possess the proper qualifications. Finally, Mr. B. E. Eisenhour of the Riverbank Laboratory (founded by Colonel Fabyan) at Geneva, Ill., obtained a magnesium alloy consisting of 95.6 per cent magnesium (one of the lightest metals known, with a specific gravity of 1.7), 0.4 per cent manganese (specific gravity, 8) and 4 per cent aluminum (with a specific gravity of 2.7). This alloy, therefore, has a specific gravity of less than 2, namely, one-third that of steel, which is usually from 7.7 to 7.9. The two great advantages of this alloy are that it is rust proof and very light.

In order to prevent tarnishing, which occurs in magnesium alloy forks, and also to prolong the period of vibration of the *c*4 (2,048 double vibrations) and the *c*5 (4,096 double vibrations) forks, further efforts were made at my instigation. By somewhat increasing the percentage of aluminum in the alloy, a new series of forks has been perfected by Mr. Eisenhour to overcome these defects. This series of forks does not discolor, but keeps its silver-like appearance, and, furthermore, the highest forks show a much longer period of vibration than did the first

series, although not quite as long as the very best steel forks. They are slightly heavier than the original forks but still have a specific gravity of about 2.8; hence they are only one-third as heavy as steel. For both air and bone conduction, they seem to serve just as well as the steel forks, and I believe that the question of obtaining tarnish-proof, rust-proof, light-weight forks at a reasonable price for all purposes of functional testing of hearing has finally been solved. When using the low-pitched steel forks, the fingers tire quite easily after making a few tests. With the magnesium alloy forks, this difficulty is eliminated. A complete range of forks extending from *C*2 (16 double vibrations) to *c*5 (4,096 double vibrations) has been perfected by Mr. Eisenhour. The price of these forks is about 25 per cent less than that of the Edelmann series.[3] With ordinary careful manipulation, these forks do not seem to break or become damaged any more easily than do the steel ones.

The constant of damping or decrement, the use of which was suggested by Harvey Fletcher for purposes of comparison with other methods, can be supplied for each of the forks at a moderate cost. By employing Fletcher's formula, one may determine the loss of hearing figured in sensation units, just as is done with the audiometer. By knowing the length of time any particular fork is heard by the normal person and by multiplying the remainder by the constant of damping, the loss of hearing is obtained in sensation units. By using a number of forks of different pitches, one obtains a curve similar to that derived with the audiometer, but it is true that doing this with many forks requires more time.

It was thought at first that owing to the light mass, the magnesium alloy forks would not be suitable for bone conduction. Action and reaction are equal, but opposite in direction, and when a heavy fork comes in contact with the skull it, so to speak, pushes the skull away, and the latter then presses against the fork with equal force. Much to my agreeable surprise, it was found that the magnesium forks served very well for testing bone conduction, even though the bone conduction is not as long as that for steel forks, especially when they are placed on the vertex.

With reference to overtones, it should be remembered that when forks are struck, not only is the fundamental sounded, but a number of overtones are produced, the first of which is very loud and has a vibration period usually five or six times that of the fundamental, and this often causes disturbing results. To eliminate overtones, weights are attached to the prongs of the forks, but these damp it, so that it

3. Edelmann, M. T.: Leitfaden der Akustik für Ohrenärzte, Berlin, S. Karger, 1911.

vibrates for a much shorter period. There are some very fine unweighted forks, such as the Bezold *A* (108 double vibrations) and *a*1 (435 double vibrations) forks, made by Edelmann, the overtones of which persist but a few seconds after they are struck. Bezold[4] himself has called attention to the fact that if weights are not provided, the overtones may be eliminated by placing a wide rubber band about each prong. This does not add materially to the weight of the fork, and only slightly shortens the period of vibration. When the unweighted magnesium forks are struck, they usually give forth rather shrill and persisting overtones, and this has been commented on by a number of men to whom these forks have been demonstrated. However, if the weights are removed from the medium-pitched Edelmann forks, it will be found that they also have disagreeable overtones. By merely slipping the aforementioned piece of rubber tubing on the prongs these overtones are easily obviated. Mr. Eisenhour has now perfected medium-pitched, unweighted forks which are practically free from overtones, the overtones lasting about one or two seconds. With the Edelmann series of forks, by the use of weights and with markings on the prongs, it is possible to set the weights at various points and thus get five or six pitches from one fork. The three lowest pitched magnesium forks are also supplied with weights at various points and thus a whole octave is obtained from one fork. This is not only a convenient but an economical provision, as it saves at least one or two forks in the series when an attempt is made to get a continuous range for the lower octaves.

Quite recently the late Prof. Karl L. Schäfer and Mr. Wetlow of Berlin presented a sounding rod of the pitch of *c*5 (4,096 double vibrations).[5] Even with the finest steel forks of the same pitch, it is usually impossible to get a vibration period longer than from twenty-five to thirty seconds. With this rod they obtained an excellent tone lasting as long as from sixty to seventy seconds. Mr. Eisenhour has devised a sounding rod of this pitch of the same material as that used in the latest model of magnesium-aluminum alloy forks, and this vibrates for from seventy to ninety seconds. He has further perfected for me a rod of the pitch of *c*6 (8,192 double vibrations) the sound of which lasts for about fifteen seconds, whereas the best forks at this pitch rarely vibrate longer than from five to eight seconds. These sounding rods are provided at their middle point with a brass grip, and when struck on either end give forth longitudinal vibrations of the pitch

4. Bezold, Friedrich: Ueber die funktionelle Prüfung des menschlichen Gehörorgans, Munich, J. F. Bergmann, 1897; Lehrbuch der Ohrenheilkunde für Aerzte und Studierende, Munich, J. F. Bergmann, 1906.

5. Schäfer, Karl L.: Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **3**:132, 1910.

for which they are constructed. The rods were demonstrated at the meeting of the American Academy of Ophthalmology and Otolaryngology held in Chicago from Oct. 27 to 31, 1930. Mr. Eisenhour is now perfecting rods up to 15,000 double vibrations.

In view of the fact that with early changes in the auditory nerve, the hearing for high tones is usually first lost, it is important to make an accurate examination of the hearing at these pitches. Since these sounding rods vibrate much longer than the corresponding forks, I believe that a decided advance has been made in the armamentarium. It seems to me that their use should be encouraged, and I am confident that otologists will find them a most satisfactory appliance for testing the high tones. The hearing of the very highest notes can be determined with the Edelmann-Galton whistle, the Schäfer-Galton whistle or, best of all, the monochord. As previously stated, in beginning degeneration of the auditory nerve there is already some impairment for the hearing of *c*4 (2,048 double vibrations), *c*5 (4,096 double vibrations) or *c*6 (8,192 double vibrations), and such changes can easily be detected with these rods.

CONCLUSIONS

Although the voice cannot be standardized, by using an unaccentuated whisper and unaccentuated conversation with proper precautions, a practical estimate of hearing ability is obtained. This, with the history and general observation of the patient, the Weber, Schwabach and Rinne tests and examination of the ear, nose, nasopharynx and pharynx, enables one to make a diagnosis. With various audiometers, graphs are obtained, and the loss of hearing figured in sensation units is noted. This may also be done with tuning forks when the constant of damping is supplied. Recently a complete series of forks made of magnesium-aluminum alloy has been perfected. These forks are rust proof and tarnish proof, very light and cheaper than the best imported steel forks. Sounding rods made of the same alloy range from *c*5 (4,096 double vibrations) upward. The *c*5 and *c*6 rods vibrate much longer than the best steel forks of the same pitches. They are very valuable for detecting early changes in the auditory nerve.[6]

180 North Michigan Avenue.

6. Sonnenschein, Robert: Analysis of the Weber Test in One Hundred Cases, Laryngoscope **21**:660 (May) 1911; Rinne Test, Ann. Otol., Rhin. & Laryng. **25**: 455 (June) 1916; Resonators, with Special Reference to the Schäfer Apparatus, Laryngoscope **23**:602 (May) 1913; Resonators as Possible Aid in Tuning Fork Tests, Ann. Otol., Rhin. & Laryng. **31**:137 (March) 1922; Rinne Test, Laryngoscope **32**:184 (March) 1922; Tr. Am. Otol. Soc. **17**:649, 1927; **18**:89, 1928. Report of Committee on Standardization of Tuning Forks and Hearing Tests, Tr. Am. Acad. Ophth. & Otolaryng. **32**:383, 1927; **33**:491, 1928; **34**:494, 1929; **35**:496, 1930.

ABSTRACT OF DISCUSSION

DR. WALTER H. THEOBALD, Chicago: Dr. Sonnenschein presented an excellent review of the methods of testing ears by the use of tuning forks. Because no one test is complete in itself, several or all of the methods used are necessary to give an accurate evaluation of the various parts of the organ of hearing. The value and correlation of these tests depend considerably on the experience of the observer, as well as on the cooperation of the patient, since all the tests are subjective. One factor to be considered is auditory fatigue. This is especially true in conditions of nerve deafness, in which fatigue is easily produced and tends to pass off slowly. The report of the Committee from the Section of Otology of the Royal Society of Medicine states that forks with a pitch of from 128 to 256 double vibrations per second cause least fatigue, whereas higher or lower pitches cause greater fatigue.

It must not be forgotten that every test of hearing is a psychologic and to some extent a mental one, as well as a test of the ear. Not only the patient's end-organ but his emotional state, his reaction to the examiner and his experience or lack of experience in undergoing such tests come into play. It is well known that the effects of similar auditory lesions vary considerably in different persons. The power of auditory perception or "auditory intelligence," as it is termed by some writers, is important. Thus, a person with normal hearing who responds slowly to conversation will be deafer with a moderate lesion of the middle ear, whereas another person with quick auditory perception—the type, for instance, who is able to follow a lecture or a play without the slightest difficulty—will have surprising ability to hear conversation even with a greater loss of hearing as indicated by tuning forks.

Dr. Sonnenschein has presented all of the latest methods of testing ears. It may not be amiss at this time to touch on a few of the modern ideas of bone conduction. In considering the accepted tests for bone conduction, such as the Schwabach or Rinne tests, it must be borne in mind that our knowledge regarding their mechanism has not advanced much since the time of Bezold. There is a tendency to accept without qualification certain ideas regarding abnormalities with reference to bone conduction, and especially in regard to so-called lengthened bone conduction. As a matter of fact, is there such a thing as lengthened bone conduction? A simple experiment will suffice to show the fallacy of this term. If, while testing in an ordinary room, a person places a finger in the external canal and a vibrating tuning fork is placed over the mastoid of the same ear, the fork will be heard for a greater length of time than when the fork is placed on the mastoid bone with the same ear not occluded. If the same person is placed in a sound-proof room and the same two tests are carried out, it will be found that there is no difference in the time of bone conduction. Furthermore, if a patient with deafness of the middle ear is tested in a sound-proof room, it will be noted that his hearing by bone conduction is no greater than that of the normal observer. What is the explanation of these two illustrations? Simply this: Bone conduction is not a fixed value either normally or in disease, but it is greatly affected by the presence of extraneous noise. Under ordinary circumstances, the normal person who has a tuning fork vibrating on his mastoid is also constantly being bombarded by more or less noise, so that his cochlea is not able to perceive the vibrating tuning fork to its greatest extent. If this man is put in a sound-proof room, his bone conduction increases at once. The person with otosclerosis with a fairly healthy inner ear has the greater part of extraneous noise shut out by virtue of the disease of the middle ear, so that his cochlea can concentrate, so to speak, on

the tuning fork which is in contact with his mastoid, thus giving a so-called lengthened bone conduction. In a sound-proof room, it will be found that his bone conduction is the same as it was under ordinary conditions, but the discrepancy between him and the normal person in the sound-proof room has disappeared owing to the latter's ability to hear by bone conduction under this noiseless environment.

DR. HORACE NEWHART, Minneapolis: There is a growing recognition of the need for greater accuracy in making hearing tests for the following purposes: (1) for research; (2) to make possible the early detection of slight, often overlooked degrees of loss of hearing, many times the first indication of pathologic changes in the hearing apparatus; (3) to meet the increasing demand for the periodic survey of the hearing of large groups, especially school children for the detection of those in need of corrective treatment or special educational care; (4) to enable the otologist to determine whether a patient can advantageously use a modern aid to hearing, and which type should be prescribed.

These requirements can in part be met by the older tests. Because of the time and effort involved in their careful application, they fail to meet the practical demands of the busy practitioner. Many otologists, without entirely abandoning the classic procedures, have found the audiometer an indispensable addition to their equipment.

The periodic testing of large groups of school children by the audiometer has already been made a regular feature of the school health program in many American cities, to the great advantage of the pupil, the teacher and the taxpayer.

A most important requirement in insuring accuracy in all tests of hearing is the elimination of interfering noises. Many cases with a significant loss of hearing are not revealed by tests made in the presence of noises prevailing in the average downtown office or school building.

An absolutely sound-proof room is beyond the means of the average practicing otologist. Nevertheless, with a little ingenuity and the help of a good carpenter he can secure an acoustically treated room which will practically eliminate the most disturbing noises. This has been accomplished on several occasions by utilizing a small space less than 10 feet long and 5 feet wide. The walls are insulated with ½ inch (1.27 cm.) celotex, insulite or similar sound-absorbing material. By making a false, suspended ceiling of the same material and covering the floor with a heavy lining material and a carpet, a surprising amount of extraneous noise is eliminated and reverberations are absorbed. The door is of solid 2 inch (5 cm.) wood, covered on each side with the insulating material, and its edges are carefully weather-stripped. Such a room can be constructed for from $100 to $150.

DR. ROBERT SONNENSCHEIN, Chicago: The fatigue which Dr. Theobald mentioned is an important factor indeed. It is true that forks of medium pitch give the least fatigue. But in using the forks of high tone, if the patient no longer hears after the fork has been removed and again returned near the ear the hearing has ceased.

As far as bone conduction is concerned, only the fundamentals were discussed. The matter of bone conduction is important, and it is true that the bone conduction is different when tests are made in a noisy place and in a noise-proof room. The noises in the average room do mask the sound. However, since the tests are always done under the same conditions, the results are comparable. One can obviate at small expense a considerable portion of the noise, not only in the way Dr. Newhart has indicated, but as we have done in Chicago by having a heavy plate glass window, rubber flooring and a celotex covering for the ceiling. In that

way we do away with the echo. As Dr. Theobald has indicated, we still do not know much about the actual mechanism of bone conduction. Bezold said that there were two types of bone conduction. When one reads some of the recent theories, one finds that the old points of discussion regarding bone conduction still hold true.

As far as the examination of school children is concerned, it is important to have equipment to test large groups; the audiometer is useful. The point I wanted to stress is this: One can make the usual tests which, together with the examination of the patient and the history, will allow one to make a diagnosis just as accurate from a clinical standpoint as with the more highly refined instruments of precision. I try to impress on students the making of the simple tests, because by means of them the diagnosis is made and the prognosis known; then one knows what treatment to institute.

ROUTES OF INFECTION IN OTOGENOUS MENINGITIS

A FULMINANT CASE OF HEMATOGENOUS ORIGIN THROUGH ANOMALOUS VASCULAR ANASTOMOSES

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In recent years the pathways by which infection may pass from the middle ear to the brain have been the object of an increasing amount of attention. Especially have the investigations lately carried out by Eagleton [1] and by Friesner and Druss [2] thrown new light on a subject that had heretofore been enshrouded with obscurity.

It has long been recognized that a considerable percentage of inflammations of the middle ear lead ultimately to complications within the cranial cavity or within the adjoining large blood vessels. A large number of possible routes of infection have been established, some of which are more frequently followed than others.

In early days extension by contact infection was the only way considered, as this was the least difficult to account for. Under this concept, infection is carried immediately from the middle ear to the membrane separating it from the cranial cavity, whether this membrane is the dura or the wall of a blood vessel. The infiltrative process, pressing with increasing force, finally results in penetration of the individual tissue crevices by round cells and pus cells. Breaking-down processes within the mastoid or the formation of an extradural abscess at length causes a reaction in the dura, involving the inner as well as the outer side of the membrane; thus a subdural suppuration occurs, or what is known as pachymeningitis interna purulenta. Thrombi then form in the small veins which course through the dura, and a purulent exudate appears as the result of adhesive processes in the adjacent soft regions, followed by a diffusion of the inflammatory process, which at last becomes suppurative leptomeningitis.

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1. Eagleton, W. P.: Intradural Conditions in Relation to Rhinology and Otology, Arch. Otolaryng. **16**:203 (Aug.) 1932.

2. Friesner, I., and Druss, J. G.: Intracranial Complications of Otitic Origin, Arch. Otolaryng. **15**:356 (March) 1932.

This concept proved satisfactory until Binswanger,[3] in 1879, made a simple differentiation between (1) transition *per continuitatem* (contact infection) and (2) transition metastatically produced. But why in one case there is a continuous suppuration pushing progressively inward while in another there is no trace of micro-organisms in the form of either small or large foci he did not know, nor are physicians any wiser in this respect today.

The theory that the more virulent organisms pass quickly through preformed pathways and produce diffuse meningitis, while the less virulent ones travel more slowly by the bony route with a resultant defensive reaction on the part of the dura, was advanced by Neumann[4] in 1907. He thought that the infection has a particular affinity for certain groups of cells, which he rated, in order of frequency, as (1) the mastoid process, (2) the sigmoid fossa, (3) the tegmen tympani and the tegmen antri, (4) the pars petrosa, including the labyrinth, and (5) the posterior wall of the semicircular canal and the posterior wall of the carotid canal.

Wittmaack,[5] in an exhaustive discussion of possible routes of infection, identified seven preformed pathways, by way of (1) persisting sutures, (2) vascular anastomoses, (3) dehiscences, (4) the labyrinth, (5) the hiatus subarcuatus, (6) the facial canal and (7) the tip of the pyramid.

The accepted modern view was expressed by Fremel[6] when he defined three possible ways for infection to pass from the middle ear to the cranial cavity: (1) via the preformed way from the ear to the brain (a wall of bone, membrane and brain substance), (2) by direct continuity and (3) via the blood vessels. As Druss and Friesner[7] recently stated, infection spreads from the tympanum and the antral cavity by direct extension through the substance of the bone, with necrosis, avoiding or involving the labyrinth or the sinus in its course to the middle or posterior fossa; or it may spread by way of the dehiscences and canals for nerves, by way of the lymphatics and the blood stream, or by a combination of any of these routes.

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3. Binswanger, O.: Zur Pathogenese des Hirnabszesses, Breslau ärtzl. Ztschr. **1**:77, 1879.

4. Neumann, H.: Der otitische Kleinhirn-Abszess, Vienna, Franz Deuticke, 1907, p. 49.

5. Wittmaack, K.: Die entzündlichen Erkrankungsprozesse des Gehörorganes, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1926, vol. 12, p. 380.

6. Fremel, F.: Zur Ueberleitungsfrage entzündlicher Mittelohrerkrankungen auf die hintere Schädelgrube, Monatschr. f. Ohrenh. **59**:409, 1925.

7. Druss, J. G., and Friesner, I.: Pathways of Infection in Abscess of the Brain: Report of Five Cases, Arch. Otolaryng. **13**:532 (April) 1931.

These writers for several years carefully prepared and studied histologically a number of temporal bones illustrative of these pathways of intracranial extension. They regarded the tegmen tympani and the tegmen antri as the usual route and cited Körner [8] as having found the bone necrotic as far as the dura in thirty-seven of forty cases. On the other hand, the fact that Hajek [9] observed the spread of infection from the mucosa of the frontal and sphenoid sinuses through the diploic veins into the meninges, while the bone remained macroscopically intact, is suggestive of a potential route through these veins for otogenous infection also.

The relative frequency of these routes still remains, however, largely a matter of conjecture, despite the supreme importance of the matter from the point of view of treatment. Fremel [6] stated that the route is seldom revealed at operation because as a rule the surgical intervention has so mangled or torn the site that good material for section is rather scarce. When the infection travels by preformed ways, macroscopic evidence is practically never present and only autopsy reveals the route.

Wittmaack [5] ventured, nevertheless, to assert that the cases in which contact infection does not occur considerably outnumber those in which it does, and that in such instances the conditions for extension are such that one cannot hope to benefit the patient by operating. Extension by preformed pathways thus gains great pathologico-anatomic significance.

Although Wittmaack [5] stated that the passage of infection through abnormally persistent sutures has not been anatomically demonstrated, it is theoretically possible for the infection to pass through all the sutures which bear any relation to inflammatory conditions of the coverings of the soft parts of the middle ear. Drainage outward through the sutures has been proved in a considerable number of cases. The petrosquamous, petrotympanic and squamomastoid fissures, when they persist, are especially worthy of consideration in this connection.

Extension of infection along vascular anastomoses has come into special prominence through recent microscopic observations, and also particularly as the result of studies of pathologic-anatomic pneumatization, which throw fresh light on the development of these routes. Wittmaack [5] expressed the belief that in certain cases there exists a hyperplastic condition of the mucous membrane lining the tympanum and mastoid cells, which communicates directly through the blood vessels with the meninges. It is probable that the lymphatics also play a similar rôle in the extension of infection. In this way infection might

8. Körner, O.: Die otitischen Erkrankungen des Hirns, der Hirnhäute und des Blutleiters, Munich, J. F. Bergmann, 1908, p. 51.

9. Hajek: Nasal Accessory Sinuses, St. Louis, C. V. Mosby Company, 1926, vol. 2, p. 620.

pass into the cranial cavity with little or no necrosis of bone, especially in acute cases.

A case has recently come under our observation which presents certain aspects of interest from this point of view.

REPORT OF CASE

J. L., a man, aged 67, was admitted to the New York Polyclinic Hospital on Feb. 17, 1933, in deep coma. Relatives stated that he had earache in the right ear two weeks prior to admission, for which he was treated by inflation of the eustachian tube. The ear did not discharge at any time, and the pain gradually abated until the day before admission, when there was a sudden recrudescence of the earache, which became intolerable. At the same time stiffness developed in the back of the neck. The condition became alarming as coma supervened, and at this juncture the patient was brought to the hospital in a critical state.

Immediately on the patient's admission paracentesis was performed on the bulging right ear drum, which elicited a large amount of blood and pus. Culture of the discharge revealed Staphylococcus aureus.

Physical examination disclosed the following: The pupils were fixed and unequal, the right one being moderately contracted. There was no reaction to light. Slight nystagmus to the left was present. The fundi were normal. The left ear was normal. The right ear drum was inflamed and bulging, with the landmarks obliterated. The mucous membrane of the nose was inflamed, and there was scant secretion. The heart and lungs were normal. There was rigidity of the muscles of the neck. Positive Babinski and Kernig signs were elicited.

The blood count showed: hemoglobin, 70 per cent; erythrocytes, 3,620,000; leukocytes, 19,000; polymorphonuclears, 92 per cent, and lymphocytes, 8 per cent. Blood culture showed Streptococcus viridans. Spinal tap on admission yielded an opaque fluid under a pressure of 44 mm. of mercury, which showed: total cell count, 4,000; white cells, 3,800; red cells, 200, and globulin, positive. Culture of the cerebrospinal fluid showed Streptococcus viridans. Spinal tap repeated at 1 p. m. the same day revealed a pressure of 36 mm. of mercury. The withdrawal of 15 cc. of fluid at this time reduced the pressure to 16 mm. The urine was normal except for a faint trace of albumin.

The temperature was 104 F. on admission and 106 F. on the following day. The pulse rate was 120 on admission and 140 on the following day. The respiratory rate was 30 on admission and 50 on the following day.

On roentgen examination the right mastoid contained a few widely scattered cells, principally in the posterior portion of the tip, anterior to the lateral sinus, extending about 1.5 cm. above the middle ear. There was a circular cavity over the lateral sinus below the knee which might be either an area of necrosis or a very large cell. The left mastoid contained a few widely scattered pneumatic cells above and posterior to the middle ear. The tip of this mastoid was sclerosed.

The diagnosis was meningitis of otitic origin. The route of infection was possibly through the diploic vessels or through the lymphatics, instead of by necrosis through the dural plate over the tympanic cavity or the mastoid antrum.

In view of the patient's advanced age, his grave physical condition and the deep coma, operative intervention was considered inadvisable. In addition, there was the difficulty of finding a localized focus of infection that could be attacked surgically.

On the following day, with a temperature of 106 F., a pulse rate of 140 and a respiratory rate of 50, the patient died.

Permission for autopsy could not be obtained.

COMMENT

In this case the primary otitis media had regressed and was apparently well on the way to resolution when, with a sudden onset, signs of fulminating meningitis appeared, with recurrence of pain in the ear and the development of the characteristic stiffness in the back of the neck. The patient died within twenty-four hours.

The markedly increased pressure of the cerebrospinal fluid, together with the opacity and the greatly increased cell count of the fluid, could mean only well established meningitis. When one adds to this the presence in the fluid of large numbers of Streptococcus viridans, there is additional evidence of generalized septic leptomeningitis.

According to Eagleton,[10] a positive blood culture is evidence of a blood stream infection, and such cultures associated with a continuously high temperature and meningeal symptoms are suggestive of (1) thrombophlebitis of the cerebral veins or (2) acute hematogenous osteomyelitis of a cartilaginous bone (the sphenoid or the petrous apex) adjacent to the meninges. Spontaneous nystagmus of cerebral origin suggests the posterior fossa as the seat of disease.

Eagleton asserted also that when meningitis develops in the course of a hematogenous infection of the mastoid, it is generally of vascular origin. The infection enters the meninges by osteothrombosis of the small vessels. After it has passed through the dura, the cortical intercerebral veins are involved. Meningitis secondary to phlebitis of the arachnoid vessels rapidly invades the base and the cortex. If it reaches the veins within the chiasmal cistern, the infected fluid is immediately disseminated to all parts of the cerebrospinal system, owing to the free communications existing between the large mesial cistern and the other cisterns of the base.

This finding of Eagleton's confirms an earlier statement by Wittmaack,[5] that a direct vascular communication of the dural periosteum with the mucosa of the middle ear exists in some persons, which accounts for the relatively frequent appearance of suppurative meningitis in infants. There seems to be no doubt that vascular anastomoses may also persist to an unusual extent in certain adults in whom there has been a standstill of development, such as occurs in the course of severe degrees of pathologic pneumatization established on a hyperplastic foundation. In such persons during the entire lifetime an abnormal

10. Eagleton, W. P.: Suppurative Meningitis of Otitic and Nasal Origin: Its Relation to Blood Stream Invasion of the Pial Vessels, Arch. Otolaryng. **15**:885 (June) 1932.

anatomic predisposition exists for the extension of inflammatory processes along these preformed paths.

Wittmaack [5] stated that under transillumination of such abnormal ramifications it is possible to see certain localizations to which the anastomoses are particularly attached. The most important one lies in the tympanic roof in the prolongation of the semicanal for the nervi petrosi. On the lower surface of the pyramid there are two such anastomoses in the neighborhood of the sigmoid sinus, one below the corner of the pyramid and the other at the knee of the sinus. It has been possible through anatomic studies to trace an inflammatory process from the mucosa of the middle ear, that is, from the mastoid cells, along vascular routes of this kind. So it appears that special significance should be attached to these ways of extension.

The kind of complication depends on (1) which vascular communication serves as a route of extension and (2) the way the process extends after it has entered the cranial cavity. Thus one may see meningitis or an abscess develop along a vascular cord communication at the roof of the tympanum, while if the process is along the posterior surface of the pyramid one may often see instead a sinus complication, but sometimes meningitis or both conditions. Hence one must consider the possibility of both types occurring simultaneously, with consequent double complications in the middle and the posterior cranial fossa. Moreover, on the floor of the tympanum wide infiltrations, with an unusually wide-meshed vascular network, are found in cases in which a hyperplastic mucosa has persisted, which is frequently infiltrated by a vascular net with especially wide lumens. Thus again one sees the special anatomic dispositions for the passage of inflammatory processes through the tympanic floor to the adjacent wall of the jugular bulb lying beneath it, in which instance they usually cause thrombosis.

It is Eagleton's [11] view that the pia-arachnoid contains the protective mechanism of the brain against infection, and that this membrane is similar to the peritoneum in structure and function; that the anatomic structure of the arachnoid spaces, with their small meshes and larger basic cisterns, is well adapted for the purpose, and that the arachnoid, which has been shown by Weed [12] to be of lower genetic origin (mesenchymal) and hence capable of rapid proliferation, is thus enabled to wall off infection. This power of resistance on the part of the arachnoid was emphasized by Ruedi,[13] who pointed out the relative infrequency

11. Eagleton, W. P.: Traumatism of the Frontal and Temporal Regions and Their Relation to Meningitis from the Standpoint of the General Surgeon, New York State J. Med. **32**:947 (Aug. 15) 1932.

12. Weed, L. H.: Dual Source of Cerebrospinal Fluid, J. M. Research **26**:21, 1914.

13. Reudi, L.: Der otogene Subduralabszess, Monatschr. f. Ohrenh. **64**:3, 1930.

of subdural collections of pus, owing to the anatomic relations of the meninges and the comparative bloodlessness of the arachnoid.

From a surgical point of view, Eagleton [14] recognized two types of meningitis, depending (1) on the mode of entrance into the meninges and (2) on the pathway by which the infection extends. He termed these two types (1) "subarachnoid space meningitis," which originates by direct extension from a primary focus of suppuration in the ear or the nose and (2) "arachnoid vessel meningitis," which is secondary to a septic infection of the blood vessels of the arachnoid.

The first type is curable by surgical methods, unless the vessels also have been attacked. Success depends on drainage of the infected areas while the infected fluid is still localized in a cistern adjacent to the primary focus. In the second type, the inflammation enters the meninges by the blood vessels and is carried by them to different parts of the arachnoid. Here the inflammation quickly becomes diffuse and forms the basis for just such a fulminant case as the one we have described, in which the sudden onset points to a process that was well advanced through blood stream infection before any signs of cerebral involvement appeared.

It is worthy of note that the micro-organism obtained on culture from the aural discharge was Staphylococcus aureus, while that in the blood and the cerebrospinal fluid was Streptococcus viridans. How are we to account for this discrepancy and for the absence of staphylococci from the blood and fluid?

Eagleton, on the basis of repeated observations at autopsy, recently expressed an interesting view that even if otitis is present at the time of an attack of meningitis, an unsuspected suppuration of the sphenoid sinus or osteomyelitis of the base of the sphenoid may be the true cause of the meningitis, the suppurative otitis or mastoiditis being only a concomitant infection secondary to the sphenoid disease. According to this author a positive blood culture appearing coincidentally with pneumococcic or streptococcic meningitis, without thrombophlebitis of a large venous sinus, calls for a careful retaking of the history, to ascertain if the initial headache and vomiting did not occur previous to all aural symptoms. Disease of the sphenoid sinus was present in twenty-five, or 40 per cent. of fifty-eight cases of meningitis with autopsy in which the state of the sphenoid was recorded, and in all of which during life the meningitis was thought to be of aural origin. The fact that in the case reported here the nasal mucous membrane was inflamed may possibly be of some interest in connection with the source and route of the infection.

14. Eagleton, W. P.: Otitic Meningitis, J. A. M. A. **87**:1544 (Nov. 6) 1926.

CONCLUSIONS

1. Various pathways exist by which infection may pass from the middle ear to the brain.

2. Extension by way of the blood vessels or lymphatics leads rapidly to a diffused meningitis.

3. Positive blood cultures are indicative of a hematogenous infection of the brain.

4. A case of fulminant meningitis is described in which the findings pointed to the blood stream as the route by which the meninges had been infected, possibly through abnormal vascular anastomoses.

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ETIOLOGY AND NATURE OF CHRONIC HYPERPLASTIC SINUSITIS

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NEW YORK

In studying the literature dealing with chronic hyperplastic sinusitis one is struck with the fact that there is no accepted consensus as to its etiology or its relationship to the allergic diseases. For the past three years we have been studying a group of cases showing various degrees of hyperplasia of the membranes lining the paranasal sinuses and covering the turbinates. These hyperplasias were found to be associated mainly with manifestations of asthma and hay fever, but various other allergic conditions have been present to a lesser extent. We think that it is essential to the proper conception of these problems to determine whether an individual case is of the skin-sensitive type or the bacterial or infective type, or belongs to both types. So many reports based on clinical or operative studies are confusing because of lack of information of this sort.

Two schools of thought have developed which seek to explain the etiology of hyperplastic sinusitis. One maintains that the allergens are the primary factors, and that the infection is a secondary development. The other maintains that the hyperplastic changes are due to a primary bacterial infection. It is difficult to prove that either theory is absolutely correct, but we shall present data which convince us that the evidence favors infection as being the true cause of the hyperplasia, and that the reaction to the bacteria is allergic on account of the constitutional tendency of the patient.

Hansel,[1] in an extensive study, both clinical and histopathologic, of the allergic conditions occurring in the nose and sinuses adhered to the theory that the inhalant and food allergens are the principal etiologic factors. He based his convictions mainly on the histopathologic findings (eosinophilic cells in the membranes, nasal secretions and blood)

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Read before the Section of Medicine of the New York Academy of Medicine, Jan. 19, 1932, and before the Society for the Study of Asthma and Allied Conditions, Washington, D. C., May 6, 1933.

1. Hansel, F. K.: Nose and Sinuses in Allergy, J. Allergy **1**:43, 1929.

and a general review of the literature. His sixth conclusion stated that "the part played by bacteria as etiologic factors in nasal allergy has not been proved, and the general consensus of opinion is that bacteria are secondary invaders." Piness and Miller [2] also expressed the belief that the bacterial infection is purely coincidental or secondary to an already existing allergic condition which causes changes in the sinus membranes favorable to bacterial growth. They asserted that "the sinusitis is the result of allergic disease but never the cause." Selfridge,[3] Todd [4] and Dean [5] have all emphasized the importance of allergic factors, Dean being one of the foremost exponents of their importance in sinusitis in children.

Goodale [6] and Kistner,[7] however, were inclined to believe that bacterial infection plays an important rôle, Kistner stating that "hyperplastic sinusitis is the latent stage of sinus infection, that in its early stages it is usually purulent, but that it may be the result of an attenuated or low grade infection on an allergic basis without ever showing evidence of purulency."

In attempting to develop this theory of bacterial allergy as the cause of the hyperplastic sinusitis, we have studied eighty cases of hyperplasia clinically, bacteriologically and pathologically. An accurate history of the age and the character of the onset of the allergic symptoms, the associated allergies, present or past, the previous antecedent allergies, complete cutaneous sensitivity tests, examination of the nose and the throat, including roentgenograms of the sinuses, and a general medical check-up constituted one part of the study. The second part consisted of: cytologic examination of the blood, the sputum and any nasal discharge or washings from the sinus; a culture of the washings and of the membranes removed later at operation, and a pathologic examination of these membranes, including special studies for bacteria, the use of autogenous vaccines with close observations on their effects on the allergic symptoms and the nasal and sinus membranes, and, lastly, the operative effect on the patient's condition after the removal of the membranes.

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2. Piness, G., and Miller, H.: Specific Protein Reactions in Eye, Ear, Nose and Throat, Ann. Otol., Rhin. & Laryng. **38**:691, 1929.

3. Selfridge, G.: Disturbances of Metabolism and Its Relation to Certain Diseases of the Respiratory Tract, Laryngoscope **38**:243, 1922.

4. Todd, F. C.: Ethmoiditis as a Common Sequel (Never the Cause) of Pollinosis, J. A. M. A. **59**:109 (Sept. 21) 1912.

5. Dean L. W.: Nasal Sinus Infection in Children, J. A. M. A. **93**:838, (Sept. 14) 1929.

6. Goodale, J. L.: Vasomotor Disturbance of Upper Air Passages and Sinus Disease, Ann. Otol., Rhin. & Laryng. **31**:882, 1922.

7. Kistner, F. B.: Histopathology and Bacteriology of Sinusitis, with Comments on Postoperative Repair, Arch. Otolaryng. **13**:225 (Feb.) 1931.

The technic of the studies of the sinuses consisted in puncturing the inferior meatal wall with a Coakley trocar, flushing sterile physiologic solution of sodium chloride into the antrum and catching the return flow in a sterile bottle. Swab cultures were also made from the ethmoids and washings taken from the sphenoid and the frontal sinuses. The washings were then examined cytologically, especially for eosinophils, and cultured on blood agar plates and in beef infusion broth. Some of the cultures were taken directly from the antral membrane when the sinus was opened at operation. After the tissues were removed, they were placed in sterile glycerin and taken immediately to the laboratory,[8] where they were washed in 95 per cent alcohol, flamed and ground in a sterile mortar, and the material was cultured as aforementioned. These bacterial procedures were carried out in a draft-free room under a hood. Cultures were examined at the end of twelve, twenty-four, forty-eight and seventy-two hours. In some of our cases cultures were made as many as eight times, and in none less than three times. In a great many instances cultures were made during acute exacerbations of the sinusitis and the asthma. The remaining tissue was sent to the pathologic department, where a microscopic examination was made and special staining for bacteria carried out, the methylthionine chloride, U. S. P. (methylene blue), and Gram technic being used.

The infective character of the membranes was clearly demonstrated by our bacteriologic and histopathologic studies. We were greatly surprised to find that all of the twenty-nine antral membranes except one gave a definite growth of one or more organisms. Of the fifty-one ethmoid, sphenoid and turbinal membranes, sixteen yielded negative cultures. The negative cultures were obtained from mucous or cystic polypi, the contents of which are frequently sterile, the infection being in the underlying membrane or cyst wall. Mullin,[9] in a recent discussion of allergy and otolaryngology, demonstrated this clearly in the case which he cited. The ethmoid and sphenoid cultures were frequently different from those obtained from the antral membrane, and the two antrums sometimes gave different results.

When we compared the organisms grown from the washings of the sinuses with those grown from the membranes we found that thirty-five specimens, or 43.7 per cent, were different from any ever recovered preoperatively, which in several cases extended back for from five to eight years. In forty-eight specimens, or 60 per cent, we recovered the same organism. These figures are not based on 100 per cent, as

8. The bacteriologic work was done by Miss Winifred Kelly of our office, and the pathologic work by Dr. Olcott, of the New York Hospital, and Dr. Sophian, of the Roosevelt Hospital.

9. Mullin, W. V.: The Interrelation of Allergy and Otolaryngology, Arch. Otolaryng. **15**:413 (March) 1932.

occasionally the same organism and a different one were found in a membrane. Staphylococcus aureus, especially the hemolytic type, predominated, while Streptococcus viridans, Staphylococcus albus, Streptococcus haemolyticus, Pneumococcus, Micrococcus catarrhalis and the nonhemolytic streptococcus followed in that order. Thirty-four positive cultures of membranes yielded single organisms, which seems important from the standpoint of the value of one organism as an etiologic agent in the disease as well as from the technical aspect.

Kistner,[7] in his work quoted previously, made cultures of four hundred sinus membranes and noted that a single organism was seldom found. However, he made no mention of a differentiation between the organisms present in the membranes and those in the washings. It is all very well to say that the organisms in the membranes are secondary invaders from the surface, as Hansel [1] and Piness and Milier [2] emphasized, but it is difficult for us to believe that such is the case after a careful study of our findings. Ashley and Frick,[10] who studied the maxillary antrums of eighty-five children bacteriologically and cytologically, and Balmer [11] stated that bacteria may be found in the membranes of an antrum giving a negative result on culture of the washings, but they added that the bacteria in most cases are secondary factors in the etiology of chronic sinusitis, an opinion which we hesitate to accept, both because of our own observations and because we feel that many of the bacteriologic studies on sinusitis are not comprehensive enough to be conclusive. Apparently a culture made from the surface of the sinus membrane cannot always be accepted as an index of the basic infection, an index which Polvogt and Crowe [12] showed could usually be accepted when one is dealing with tonsils.

After the bacteria were frequently recovered in these hyperplastic membranes by cultural methods, we studied the tissues pathologically, with special reference to staining of the bacteria in the tissues. In over 85 per cent of the specimens examined, organisms were definitely demonstrated, while in the others the results were too questionable to be considered positive. It is interesting to note that our one culturally negative antral membrane had cocci in the tissues. Kistner [7] reported that he was able to demonstrate the organism in the tissue microscopically as frequently as by culture. He quoted Rosenow, to whom

10. Ashley, B. J., and Frick, W. V.: A Bacteriologic and Cytologic Study of the Maxillary Antrum in Children, with a Clinical Study of 83 Cases, Ann. Otol., Rhin. & Laryng. **39**:605, 1930.

11. Balmer, F. B.: The Relation of Clinical to Bacteriologic Observation in Normal and in Diseased Maxillary Antrums, Arch. Otolaryng. **14**:440 (Oct.) 1931.

12. Polvogt, L. M., and Crowe, S. J.: Predominating Organisms Found in Cultures from Tonsils and Adenoids; Observations After 100 Operations, J. A. M. A. **92**:962 (March 23) 1929.

he sent specimens for examination, to the effect that the organisms were most frequent in the deeper layers of the membrane close to the periosteum, becoming fewer toward the surface or epithelial layer. It is our opinion that the hyperplastic changes are an allergic response of a specialized tissue membrane to these bacteria or their products, resulting from low grade infections in a patient with a peculiar constitutional allergic capacity.

Our general pathologic examinations only verified what has long been known to characterize the hyperplastic membranes; namely, that the epithelium is thickened, often infolding and polypoid, with desquamation of the cilia, a thickened hyalinized basement membrane and, in the tunica propria, marked edema, glandular hyperplasia, proliferation of the connective tissue and eosinophilic, round and plasma cell infiltration. Neutrophils are rare in these chronic cases. The importance of this thickened basement membrane and the predominance of eosinophils in the membrane of allergic persons has been emphasized by Shambaugh [13] and by Coates and Ersner.[14] Huber and Koessler,[15] in 1922, reported eosinophilic infiltration in the bronchial membrane of asthmatic patients who came to autopsy. This is certainly the same process that is going on in the sinus membrane, and, as one of us (R. A. C.[16]) has reported recently in another paper, the eosinophilia "may be regarded as a bacterial sensitization phenomenon equally as well as it is so regarded in serum disease and experimental anaphylaxis."

In our clinical studies of the hyperplasias we have checked a large series of true cases of pollen hay fever and of vasomotor rhinitis. It is unusual to find nasal polypi or even chronic thickening of the turbinate membrane in the absence of complicating inflammatory processes. On the other hand, in several cases which have been followed for a number of years and in which roentgenograms have frequently been made we have watched the hyperplasia develop with infection. In a series of one hundred and seventy-five of these infective allergic cases, 48.3 per cent showed a definite polypus formation from the sinuses. We do not here include the irregular polypoid thickening of the turbinates.

During the past three years we have also collected considerable data which assist in establishing the allergic character of infective hyperplastic membranes. One of us (R. A. C.[16]) has presented previously a

13. Shambaugh, G. E., Jr.: The Basement Membrane in the Mucosa of the Upper Respiratory Passages, Arch. Otolaryng. **13**:556 (April) 1931.

14. Coates, G. M., and Ersner, M. S.: Occurrence of Eosinophiles in the Mucous Membrane of the Maxillary Sinus in Asthmatic Patients, Arch. Otolaryng. **2**:158 (Feb.) 1930.

15. Huber, H. L., and Koessler, K. K.: Pathology of Bronchial Asthma, Arch. Int. Med. **30**:689 (Dec.) 1922.

16. Cooke, R. A.: Infective Asthma: Indications of Its Allergic Nature, Am. J. M. Sc. **183**:309 (March) 1932.

report of studies in which it was shown that the hereditary influence is as prominent in these cases as it is in the true skin-sensitive allergic conditions. Skin-sensitive allergies are frequently present in the antecedents, while the actual hyperplastic changes have been traced through two generations in several groups which we are following. At the same time 30 per cent of these cases showed in addition an associated allergy such as hay fever, vasomotor rhinitis, urticaria, angioneurotic edema, migraine or eczema. Cutaneous sensitivity was demonstrated in almost 50 per cent of the group studied. Sensitivity to acetylsalicylic acid has occurred only in our infective cases.

In the same paper,[16] the phenomenon of eosinophils in the nasal and antral exudates, the sputum and the blood was presented, and the allergic principles involved were discussed. Those observations have been extended, and we are convinced that the presence of eosinophils in the washings from the sinuses and in the sputum is a more constant occurrence than in the blood. When these findings are compared with the negative findings in the cases of chronic suppurative sinusitis, one can readily see that we are dealing with different reactions. The theory was advanced that the eosinophilic production is a local histogenous one in the sinus and bronchial membranes and represents a local bacterial allergic reaction, with subsequent formation of eosinophils in the infected membranes and thence in their secretions. The eosinophils are not all morphologically identical with those found in the blood at the same time, some being polymorphonuclear, but most of them mononuclear and resembling both lymphoid and reticulo-endothelial cells.

We have adduced further evidence of the allergic nature of our hyperplastic cases by studying the local clinical symptoms and physical findings. The symptoms in the cases of polypi are often the same as those in cases of acute pollen-sensitive, vasomotor rhinitis, namely, sneezing, a watery discharge and an intermittent obstruction which becomes complete. The appearance of the membrane during an acute infective exacerbation is that of a pollen hay fever: it is pale, swollen, edematous and watery, in addition to being extremely sensitive to any topical application. The hyperplasia may occasionally show a superimposed allergic or vasomotor condition, but by elimination of or treatment with allergens only the vasomotor condition subsides, while the true hyperplasia due to infection remains. We have an illustrative case of this nature in a physician, tests of whose skin revealed a marked sensitivity to milk. On elimination of milk from his diet the turgescence of the membrane and the vasomotor symptoms disappeared, and he became more comfortable, but the hyperplasia remained and had to be removed operatively. Since that time he has been in excellent condition. However, in our experience, except for the inhalants, this type of association and response to treatment is rare. Forty per cent of a group

of patients with hyperplasia studied showed no immediate or delayed cutaneous reactions, and in the 60 per cent giving positive reactions we have been unable to demonstrate an appreciable effect from specific treatment as far as the hyperplasia is concerned.

Proetz [17] stated that he demonstrated with iodized poppy seed oil 40 per cent that the sinus membrane has the capacity for an allergic reaction similar to the vasomotor disturbance of the nasal membrane in hay fever. He quoted also a case of Beck's. These apparently were not cases of organically diseased membranes, judging from the reports. In our own experience with a large number of these allergic cases we have not been able to show by transillumination or roentgenograms any allergic swelling of noninfected sinus membranes during the acute exacerbations of the hay fever type. However, we believe that we have been able to demonstrate the allergic nature of diseased membranes with our autogenous vaccines. Among our patients are many who have had previous resections of the antral window, and we have been able to observe the membranes closely. In one group, 75 per cent of the patients showed a sensitivity to the vaccine, often a specificity to one organism when several were recovered. We could reproduce not only the asthma but also a local exacerbation of the sinuses with marked edematous swelling almost obliterating the antral cavity and accompanied by an outpouring of mucus or mucopus which contained numerous eosinophils. We believe that this specificity of our autogenous vaccines goes far toward proving the allergic nature of the infective membranes and toward showing that the asthma is a result of the sinusitis and not vice versa, as Schenck and Kern [18] recently stated in their paper. Our observations show that the hyperplastic changes in the sinus develop first, and that the asthma is secondary. Furthermore, to indicate that sinusitis is the cause and not the result, we have observed instances in which the membrane is removed from the sinuses and the patient is relieved of the asthmatic symptoms.

SUMMARY

1. The present theories as to the etiology of chronic hyperplastic sinusitis are those of (*a*) allergy (in the skin-sensitive type), (*b*) infection and (*c*) a combination of allergy and infection.

2. We believe that the hyperplasia is due to a primary bacterial infection in a person with an allergic constitution, and that it represents a sensitization to the infection.

17. Proetz, A. N.: Sudden Allergic Reactions in the Maxillary Sinus, J. Allergy **1**:324, 1930.

18. Schenck, H. P., and Kern, R. A.: An Evaluation of the Therapeutic Effect of the Caldwell-Luc Operation in Bronchial Asthma, J. Allergy **3**:296, 1932.

3. A group of eighty cases of hyperplasia was studied from the clinical, bacteriologic and pathologic points of view.

4. We believe that the membranes are primarily infective because (*a*) organisms are frequently demonstrated in the membranes by tissue cultures and sections, (*b*) cases of true cutaneous sensitivity without infections have not shown hyperplastic changes, and (*c*) with subcutaneous injections of autogenous vaccine one can cause a local reaction of the tissues in the sinuses.

5. We believe that the infected hyperplastic membranes are allergic because of (*a*) the frequent positive history of an allergic antecedent, (*b*) the presence of other clinical allergies in 30 per cent of the cases, (*c*) the frequency of cutaneous sensitizations, (*d*) the presence of eosinophilia in washings from the sinuses and in the membranes, sputum and blood and (*e*) the observed reaction of the membranes of the sinuses to a subcutaneous injection of autogenous vaccines.

SUPPURATION OF THE TEMPORAL BONE ACCOMPANIED BY INFECTION IN THE BLOOD STREAM

A CLINICAL STUDY

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Clinical and surgical judgment is often challenged when suitable therapeutic measures must be selected for patients with infection of the blood stream originating in the temporal bone. The attitude toward the surgical procedure to be employed is more conservative than it formerly was, and this change in view may be considered an indication of improved appreciation of the significance and importance of the various pathologic factors involved.

In otologic literature, there is a general tendency to speak of general sepsis of otitic origin as being due to sinus thrombosis, whereas in reality sinus thrombosis is only one of the ways by which infection of the blood stream may originate from infection in the temporal bone. If the nature of the pathologic conditions which cause the general septic manifestations were to be classified more accurately, evaluation of suggestions for treatment of this serious complication of mastoiditis would be less difficult. The pathologic factors have been described in a rational manner by eminent otologists. The rationale of surgical treatment is based on sound principles. The new suggestions, when carefully analyzed, usually prove to be only applications of sound surgical principles already established.

At the risk of repeating information which all modern textbooks on otology contain, it properly might be recalled that infection of the blood stream, with origin in the temporal bone, may take place by several routes or as a result of certain antecedent events. These, and measures to control the disease, are as follows:

1. Primary, direct extension into the veins of the middle ear and mastoid process at the onset of the infection in the ear. The nature of the infection is such that treatment is usually futile.

2. Direct extension of the infection of the middle ear into the jugular bulb, without evidence of surgical mastoiditis. Tying the jugular vein and blocking the bulbar end of the sigmoid sinus are indicated.

From the Section on Otolaryngology and Rhinology, the Mayo Clinic.

Read before the Section on Laryngology, Otology and Rhinology at the Eighty-Fourth Annual Session of the American Medical Association, Milwaukee, June 16, 1933.

3. Extension by osteothrombotic phlebitis, in the presence of hemorrhagic mastoiditis. The symptoms may be present throughout the course of the disease and subside when the diseased mastoid cells are completely removed.

4. As a result of the sinus phlebitis in hemorrhagic mastoiditis. This may be the advancing stage of osteothrombotic phlebitis. The wall of the sinus is definitely affected; not necessarily the intima, however. The symptoms appear suddenly in the course of the disease after a period during which the symptoms and signs have seemed negative. Complete removal of the mastoid cells and exposure of the wall of the sinus, even though the sinus plate may appear normal, are indicated. The area of phlebitis may be situated at any place on the surface of the sigmoid sinus.

5. As a result of mural thrombosis of the wall of the sinus, occurring as an advanced stage of osteothrombotic phlebitis, or of phlebitis of the wall of the sinus in the hemorrhagic type of mastoiditis, or as the extension of the infection through the wall of the sinus in the coalescent suppurative type of mastoiditis. Depending on the general physical condition of the patient, the surgical indications are removal of the thrombus and obliteration of the sinus; in addition, it may be advisable to tie the jugular vein.

6. Extension of the infection owing to breaking down of an obliterating thrombus. This occurs usually late in the course of the coalescent, suppurative type of the disease. Removal of the thrombus, obliteration of the sinus and, depending on the general physical condition of the patient, tying of the jugular vein are the surgical indications. In certain cases, removal of the clot and temporary closing off of the sinus are effective. Under these circumstances, the sinus may be expected to function again.

7. As a result of infected thrombosis of the jugular vein. The surgical indication is removal of the vein below the extent of the thrombosis.

8. As a result of infected thrombosis of the petrosal sinuses. Surgical treatment, even if the diagnosis has been made, would be extremely difficult.

The surgeon's responsibility in operating is not terminated until sufficient pathologic change to account for the clinical signs and symptoms has been encountered. It may be safer, when in grave doubt, to tie the jugular vein than not to do so.

PURPOSE AND MATERIAL

The series of cases to be presented here were studied to evaluate (1) the operative procedures that were deemed necessary to control

the type of disease that was encountered, and (2) the effect of the surgical treatment that was adopted.

The series includes fifty-two cases, each of which represented one or more of the following: definite sinus phlebitis, mural thrombosis, obliterating thrombosis of the sigmoid sinus or lateral sinus and thrombosis of the jugular vein. No cases of primary thrombosis of the jugular bulb were encountered. Cases in which a septic type of temperature occurred early in the course of hemorrhagic mastoiditis and in which the disease was controlled by complete operation on the mastoid, without exposure of the wall of the sinus, were not included in this study.

The cases were further divided into the following classes: (1) those in which the operative intervention for infection of the blood stream was a primary procedure, and (2) those in which the operative intervention for the infection of the blood stream was secondary to the original operation on the mastoid process. Diagnostic features of infection of the blood stream following infection of the temporal bone cannot be considered here.

PRIMARY OPERATIVE INTERVENTION FOR INFECTION OF THE BLOOD STREAM

Phlebitis of the Sigmoid Sinus in Hemorrhagic Mastoiditis.—In my earlier surgical experience with suppurative disease of the mastoid process, I was impressed by the fact that pathologic changes might not be found within the sinus when it was explored because of the presence of symptoms of infection of the blood stream, even though the outer surface might be covered with granulations. This was especially noticeable in the hemorrhagic type of disease. Also, it was observed that when the sinus plate appeared to be normal, it was, nevertheless, frequently adherent to the wall of the sinus. When the plate was removed, however, the sinus was not normal in color, but perhaps was pink and studded with small bleeding points, and the surface was moist. In other cases there were definitely localized, red, raised areas. It was found, too, that even though the middle portion of the sigmoid sinus was exposed and was found normal, further exploration disclosed involved portions at the upper or lower turns of the sinus. For this reason I now explore further as a routine, and in addition to complete removal of the mastoid cells, the wall of the sinus is exposed. In my experience sinus phlebitis has affected young persons for the most part. The symptoms suggestive of invasion of the blood stream have appeared suddenly in the course of the disease.

The anatomic type of mastoid process seems to be of significance. The disease has been encountered most often in the presence of the

diploic and small cell type of mastoid process. Clinically, it is known that in such an anatomic type of mastoid process, the acute inflammations are more frequently associated with exaggerated pain and fever.

Blood cultures and estimations of hemoglobin are extremely important aids in determining progress in this disease. The general appearance, too often not taken into consideration, is a fairly good index of the actual general state of the patient. Blood transfusion, when indicated, may be used as a supportive measure. Striking clinical effects are commonly produced.

Seventeen cases of sinus phlebitis were reviewed for this study. The sinus plate appeared to be normal in sixteen. Eleven patients were less than 12 years of age, four were less than 20 and two were more than 20. The average time of onset of the symptoms of invasion of the blood stream was twelve and six-tenths days after the onset of mastoiditis. The earliest appearance of the symptoms was on the third day of mastoiditis, and the latest on the thirty-sixth day.

One large cell variety of mastoid was encountered. The sinus plate was normal, but on the wall of the sinus there were definite physical changes which were thought to account for the clinical symptoms and signs. Five of the seventeen patients had bilateral involvement of the mastoid processes, for which bilateral mastoidectomy was performed. In two of these cases, bilateral mastoidectomy was performed originally, and sinus phlebitis was encountered on both sides. In another case, a year elapsed after unilateral mastoidectomy before the onset of infection in the opposite ear was followed by the same chain of symptoms, and operation revealed phlebitis of the sigmoid sinus. In the remaining two cases of the five, operation on one side was followed, after an interval, by operation on the other side, because the patients were not convalescing satisfactorily. The process on the side on which the later operation was performed seemed, from physical appearance, to be resolving well. Nevertheless, it was assumed that pathologic conditions still present might be causing the existent trouble. That the assumption was justified was apparent when definite sinus phlebitis was revealed on the side of apparent resolution. The clinical course immediately became favorable. Four patients were given transfusions of blood. All seventeen patients recovered.

Primary Obliteration of the Sigmoid Sinus for Thrombosis.—There were eleven patients to be considered in this group (table 1), five of whom were 12 years of age or less. The oldest patient was 64 years of age, and the youngest 3 years. The general condition of the patients was such that it was felt that if they did not respond to the more conservative operation, the jugular vein could be attacked later.

Obliterating thrombi were encountered in four cases, and free bleeding was obtained from both bulbar and torcular ends in three. In the

one case in which there was no bleeding from the torcular end, the sinus was uncovered to the torcula, where it was found that the thrombus extended into the longitudinal (superior sagittal) sinus. Convalescence was protracted. In one of the four cases pyelitis developed, but responded to treatment. Otherwise, convalescence of these four patients was uneventful.

TABLE 1.—*Primary Obliteration of the Sigmoid Sinus for Thrombosis in Eleven Cases*

Pathologic Condition Found at Operation	Cases	Source of Bleeding After Sinus Was Opened: Torcula Herophili	Source of Bleeding After Sinus Was Opened: Jugular Bulb	Postoperative Course: Events	Postoperative Course: Cases	Further Operations	Results
Obliterating thrombus	4	3	4	Normal course	2	None	Good
				Pyelitis	1	None	Good
				Extension to longitudinal sinus	1	None	Slow convalescence
Mural thrombosis	4	4	4	Negative	3	.....	Good
				Septic fever	1	Ligation of jugular vein and transfusion on third postoperative day	Good
Broken-down thrombus	1	0	1	Fever	1	Ligation of jugular vein on third postoperative day and transfusion on seventh postoperative day	No effect from operation but patient decidedly improved
Phlebitis (hemorrhagic)	1	1	1	Fever and chills	1	Ligation of jugular vein on third postoperative day and transfusion on seventh postoperative day	Little effect from operation but patient decidedly improved
None	1	1	1	Fever and chills	1	Ligation of jugular vein on third postoperative day	Cavernous sinus thrombosis developed; death on tenth postoperative day; necropsy disclosed thrombosis of petrosal sinuses

Mural thrombosis was encountered in four cases, and there was good bleeding from both ends of the sinus. Convalescence was without incident in three of these cases. In the fourth case it was apparent that the clinical course was not favorably affected. Pleurisy and an infection in the ankle developed on the second day following operation. On the third day, the jugular vein was ligated and divided, and at the same time a transfusion of 500 cc. of citrated blood was given. In the blood culture hemolytic streptococci made very active growth.

In one case a broken-down thrombus and pure pus were encountered within the sinus. The end toward the bulb bled freely, but no

bleeding was obtained from the end toward the torcula. The clinical course was not favorably affected; therefore, on the third day after obliteration of the sinus, the jugular vein was ligated and divided. This procedure had little effect. On the seventh day, transfusion of 500 cc. of citrated blood was given, and the effect was immediately good. Whether it might have been better judgment to have tied the vein at the first operation is a question. The transfusion had more effect clinically.

The patient with phlebitis was encountered while an epidemic of influenza was in progress. Streptococcus viridans grew in the blood culture. Because mural or obliterating thrombosis was not encountered, it was thought that obliteration of the sinus would be sufficient. This case, in fact, was the one that caused me to change my ideas about the surgical care of this type of disease. The clinical course was not satisfactory; therefore, the jugular vein was tied and divided on the seventh day after obliteration of the sinus. This had no apparent effect. This case was the first in which I employed transfusion of blood as a supportive measure. The result was strikingly good.

The remaining patient of the eleven had been sick for six weeks, and because of the negative findings in the sigmoid sinus, the jugular vein was nof ligated. Symptoms of cavernous sinus thrombosis developed on the second day, and death occurred on the tenth day. Necropsy revealed thrombosis of the petrosal sinus. It is problematic whether primary ligation of the jugular vein would have affected the course favorably.

It is seen that of the eleven cases in which primary obliteration of the sigmoid sinus was carried out, the jugular vein was subsequently ligated in four. However, transfusion seemed to have a more beneficial effect than ligation of the jugular vein. Because ligation of the jugular vein is an accepted form of treatment, its judicial use might avoid medicolegal complications.

Primary Obliteration of the Sigmoid Sinus and Primary Jugular Ligation.—The five patients in this group presented symptoms of sepsis of sufficient severity to warrant primary operation on both the sigmoid sinus and the jugular vein. Two of the patients were less than 12 years of age, and the other three were less than 30. Three had had chronic suppurative otitis media of many years' duration.

The vein was ligated and divided as the first step in the operation in two of the five cases, and in one of these thrombosis of the jugular vein was found to extend to the clavicle. In this instance, the vein was resected. In the other three cases of the five, the vein was ligated and divided because bleeding was not obtained from the bulbar end of the sinus. Parasinuous abscess, with necrosis of the wall of the sinus and

obliterating thrombosis, was found in four of the cases. In one case in which symptoms had been present for six days, the thrombosis was of the mural type.

Ordinarily, in otologic practice, operations of this type are considered emergency procedures. In this group of five cases only one operation was performed as an emergency and that was because the medical attendant urged it. It is my belief that a good rest is more beneficial than harmful for patients who may have come from a considerable distance. Also, the interim gives ample opportunity to complete physical and laboratory examinations so necessary in the estimation of the real condition.

Transfusion of citrated blood was used in three of the cases, one preoperatively and two postoperatively. One patient continued to have a septic type of temperature for two weeks. During this time, the wound in the neck became infected, and an abscess developed over the sternoclavicular joint, owing to spread of the infection within the sheath of the sternocleidomastoid muscle. All the patients in this group recovered.

SECONDARY OPERATIVE INTERVENTION FOR INFECTION OF THE BLOOD STREAM

Invasion of the Blood Stream After Operations Performed at the Clinic.—There were fourteen patients in this group (table 2). Six were less than 12 years old, and the ages of the remainder ranged from 18 to 64 years. The question arises whether, in this group of cases, the pathologic changes were present at the time of the original operation and remained unrecognized, or whether the operative intervention was not extended far enough to disclose significant signs. In a case in which the symptoms develop immediately after operation, the probabilities are greater that the pathologic changes which caused the symptoms were present at the time of the operation on the mastoid. This would be particularly true if all operations on the mastoid were performed as emergency procedures in those cases in which the surgeon had not observed the patient long enough to suspect invasion of the blood stream. Being forewarned is to be forearmed. In cases 3, 6, 8, 9, 10 and 14 (table 2) symptoms developed soon after operation.

In case 3 acute follicular tonsillitis developed following the operation, and this syndrome masked the symptoms of extension of the infection from the ear. As the tonsillitis cleared, the symptoms did not. In case 6, if the thrombus in the bulb was present at the original operation, it was not recognized. In case 8, the probabilities are that pathologic conditions were present, although the preoperative course did not suggest invasion of the blood stream. In case 9, exploration of the sinus and vein gave negative results. Neurologic signs suggestive of

TABLE 2.—*Cases in Which There was Invasion of the Blood Stream Following Operation on the Mastoid Process Performed at the Clinic*

Case	Time of Onset After Mastoidectomy, Days	Operation	Operative Findings	Postoperative Course	Result	Comment
1	15	Obliteration and ligation*	Thrombus in sigmoid sinus	Two transfusions	Good	
2	14	Obliteration and ligation	Thrombus in sigmoid sinus; jugular vein collapsed	Meningitis	Died on seventh day after sinus operation	Left town on ninth day after mastoidectomy against advice; returned in a month
3	4	Obliteration and ligation	Thrombus in sigmoid sinus; jugular vein collapsed	Air embolism	Good	Tonsillitis obscured picture at onset
4	14	Obliteration and ligation	Negative	Pneumonia and nephritis	Died on twentieth day after mastoidectomy	Pneumonia probably the cause of symptoms
5	7	Obliteration	Mural and emissary vein thrombosis	.....	Good	Wound healed slowly
6	4	Obliteration and ligation	Thrombus extending into jugular bulb	Pulmonary suppuration; spontaneous evacuation 250 cc. of pus	Good	Transfusion with improvement; protracted course until evacuation of pus
7	7	Obliteration and ligation	Peribulbar abscess; obliterating thrombus; jugular vein collapsed	.....	Good	Never appeared very ill
8	3	Obliteration; resection on tenth day after mastoidectomy	Sigmoid sinus and jugular vein thrombosed	Pneumonia on third day after obliteration and ligation; empyema; choked disks	Died on fifteenth day after mastoidectomy	Should have been operated on earlier in course
9	2	Obliteration and ligation	Sinus and vein normal	Transfusion; septic throat; hemiplegia and aphasia	Died on thirteenth day after mastoidectomy	Intracranial symptoms; source not determined; no necropsy
10	..	Obliteration	Thrombus in lateral sinus	Jugular bulb injured at time of operation on labyrinth	Good	Injury of jugular bulb does not account for thrombus in lateral sinus
11	6	Obliteration and ligation	Obliterating thrombus in lateral sinus; jugular vein collapsed	Transfusion on second postoperative day	Good	
12	9	Obliteration on tenth day after mastoidectomy; ligation on seventeenth and resection on twenty-fourth	Bulb and sigmoid sinus thrombosed	Septic focus in sacro-iliac joint	Good	Thrombosis of jugular vein the important factor
13	6	Obliteration and ligation on twenty-second day after mastoidectomy	Thrombus of sigmoid sinus; jugular vein collapsed	Erysipelas on sixth day after mastoidectomy	Died	Cerebrospinal meningitis
14	4	Obliteration and resection	Sinus normal and jugular vein thrombosed	Transfusion	Good	Sinus normal; thrombus in vein

* Obliteration of sigmoid sinus and ligation and resection (when done) of jugular vein.

different pathologic processes were present. In case 10 there was a typical clinical picture. The bulb was injured during a radical operation, but the thrombus was found high in the sigmoid sinus. The obliteration of the sinus was effective. In case 14, I am convinced the thrombus was present in the jugular vein at the time of the operation on the mastoid, but because the parasinuous abscess seemed sufficient to explain the clinical course, the vein was not explored. At the time of the secondary operation, the sinus was found to be normal, but a definite thrombus was encountered in the jugular vein. The significance of thrombosis in the jugular vein is shown in cases 12 and 14, which seem to refute the argument that the jugular vein need never be subjected to surgical intervention. It will be recognized that erysipelas might offer many difficulties in the evaluation of symptoms suggestive of invasion of the blood stream, as in case 13.

In those cases in which the symptoms of invasion of the blood stream develop after ten days or two weeks, it is not so probable that the pathologic conditions causing the invasion were overlooked at the original operation on the mastoid. Many patients were dismissed from the hospital because their convalescence had apparently been satisfactory. This obtained in cases 1, 2 and 4 in this group. In case 2 the patient was taken from the city against advice and was returned two weeks after the symptoms of infection of the blood stream had become manifest. At that time signs of meningeal involvement were present, and the patient appeared to be moribund. Operative intervention had no beneficial effect. In case 4, the symptoms which developed on the fourteenth day were suggestive of pulmonary infection, but the clinical signs in the thorax were said to be negative; for that reason, the sinus and jugular vein were explored. The findings were negative, but pneumonia developed immediately, followed by empyema, and death occurred on the twentieth day. It is true that pneumonia may develop in the convalescent period following any operation, but following an operation on the mastoid, the suspicion of invasion of the blood stream from the operative field is uppermost always.

In the fatal cases, it would be difficult to say whether different treatment would have prevented death, except in cases 2 and 8. In case 2 the neglect was due to the ignorance or indifference of the parents, and in case 5 the clinical picture was masked by signs of meningeal and thoracic involvement. The patient in case 13 was an old man who had neglected the original condition of the ear, although he was warned, and erysipelas developed six days following operation on the mastoid. Whether invasion of the blood stream developed at the same time was not determined.

A review of the operative findings in the postoperative group is interesting. Obliterating thrombi of the sigmoid or lateral sinuses were

encountered in ten cases, and mural thrombosis in one; the jugular vein was found collapsed above its anterior facial tributary in five. Thrombosis of the jugular vein was found in three cases.

Obliteration of the sigmoid sinus and ligation and division of the jugular vein between ligatures were performed in nine cases, resection of the jugular vein in three cases, removal of the thrombus and temporary obliteration of the sigmoid sinus in one case and permanent obliteration in one case. Transfusion of citrated blood was used in five cases.

Five patients died. This is a mortality of about 35 per cent for this group. If cases 4 and 9 are not included in the group because

TABLE 3.—*Invasion of Blood Stream Following Operation on Mastoid Process Performed Elsewhere*

Case	Operation*	Operative Findings	Postoperative Course	Comment
15	Obliteration and ligation, left side; condition unilateral	Broken-down thrombus in sigmoid sinus; jugular vein collapsed	Died on thirteenth day	Endocarditis and multiple pulmonary abscesses found at necropsy
16	Obliteration and ligation, left side; condition unilateral	Mural thrombosis of sigmoid sinus	Temperature 106 F. on fifth day; symptoms disappeared after sinus packs were removed	
17	Obliteration and ligation, left side; condition unilateral	Thrombus in sigmoid sinus; jugular vein normal	Choking of ocular disks which gradually disappeared	Sinus aspirated, which could have caused the thrombus
18	Obliteration and ligation, left side; condition unilateral	Necrotic sinus wall	Four transfusions and administration of antistreptococcus serum necessary	Convalescence stormy
19	Obliteration and ligation, left side; condition bilateral	Right jugular vein and sigmoid sinus normal; left jugular bulb and sigmoid sinus thrombosed; left jugular vein collapsed	Transfusion necessary	Staphylococci found on blood culture

* Obliteration of sigmoid sinus and ligation of jugular vein.

death was due to other causes than infection of the blood stream from a source in the temporal bone, the rate is about 21 per cent.

Invasion of the Blood Stream After Operations Performed Elsewhere.—Secondary operations on the mastoid are not always satisfactory, but if they are accompanied by signs and symptoms of invasion of the blood stream, the difficulties are increased. Case 5, in the group now under consideration (table 3), presented difficulty in the way of determining from which one of the mastoid processes that recently had been operated on the symptoms of two weeks' duration arose. This could not be determined from the clinical examination, and the Ayer-Tobey test did not help. Exploration of the vein and sinus on the right side gave negative results. The thrombus was found in the left sigmoid sinus and bulb.

The patient in case 1 (table 3) was very sick. Several operations had been performed on the mastoid, but the sigmoid sinus had not been exposed. At operation, a broken-down thrombus was encountered in the sigmoid and lateral sinus, and the jugular vein was collapsed above its anterior facial tributary. Surgical intervention had no effect. Necropsy revealed endocarditis and multiple miliary abscesses of the lung.

Whether aspiration of the sinus through its wall is a good measure is open to discussion. In case 3 this procedure was followed by sinus thrombosis. Spontaneous hemorrhage from an exposed wall of the sinus must be a rare occurrence. It occurred in case 4 following a postoperative dressing. The occurrence was followed by symptoms of sepsis, and convalescence was protracted. Four transfusions of citrated blood were used as a supportive measure. Intravenous injection of blood serum apparently produced a beneficial effect. I offer no explanation of the phenomenon. In this group of cases operative intervention consisted of obliteration of the sigmoid sinus and ligation and division of the jugular vein.

CONCLUSIONS AND SUMMARY

1. Infection of the blood stream secondary to infection in the temporal bone is serious.

2. If surgical procedures are adapted to the patient and to the source of the invasion, the favorableness of the prognosis is enhanced.

3. Obliteration of the sigmoid sinus and ligation of the jugular vein are not indicated in all cases.

4. Transfusion of blood has great usefulness as a supportive measure but does not supplant surgical removal of the infecting focus.

5. It is not easy to explain why the mortality rate is increased when infection of the blood stream follows operations on the mastoid process, unless the early diagnosis may be masked by other physical findings that could account for the clinical course.

6. Operation for relief of invasions of the blood stream associated with infection of the temporal bone need not always be considered to be of emergency expediency.

7. The mortality rate for the group studied was 13.4 per cent. If the case were eliminated in which death was caused by other conditions than infection of the blood stream from the temporal bone, the mortality rate would be 10.5 per cent.

ABSTRACT OF DISCUSSION

Dr. George M. Coates, Philadelphia: Most of us no longer feel that it is necessary in all instances to ligate and possibly to resect the jugular vein. If the occluding thrombus is located in the sigmoid portion, the bulb and vein not being

involved, ligation of the vein may well be omitted or left for a later date. If free bleeding cannot be obtained from the lower end of the opened sinus, it may be presumed, in the absence of signs of sepsis or metastasis, that the general circulation is already protected by a sterile thrombus, and ligation may be held in reserve. But when the infectious process extends into the internal jugular vein, or when serious symptoms of sepsis exist—or persist after obliteration—I feel much safer with the vein tied or excised.

It is not necessary to drain the sinus in every case showing signs of infection in the sinus. In many cases in which the interior of the sinus is not definitely infected, the infection clears up after a thorough removal of the focus of infection in the mastoid. These cases include the perisinuous or parasinuous abscesses, as well as localized areas of phlebitis, but the condition of the bony sinus plate is not always a criterion of the condition of the underlying organ. It should be removed for inspection of these structures whenever the symptoms are not sufficiently accounted for by the pathologic process in the mastoid. I agree with the essayist that when in doubt it is safer to ligate the internal jugular vein.

In Dr. Lillie's series of fifty-two cases there was no case of primary infection of the jugular bulb, which seems unusual. I have seen several, and I believe these cases are frequently among the most difficult to diagnose. In such a case ligation is indicated.

When it is at all possible, time should be taken for a thorough and complete study, and one or more preliminary transfusions given before operation.

Transfusions will benefit most patients after operation. Small amounts of blood, from 150 to 250 cc., injected at short intervals, are preferable to one large injection. Occasionally an immunized donor is available, and if so, the value of the procedure is much enhanced. Pregl's solution (the solution which results from the action of iodine on sodium carbonate solution) seemed to be of some help.

Among a thousand cases of mastoiditis in which operation was performed in my service at the Graduate Hospital of the University of Pennsylvania, fifteen were complicated by lateral sinus thrombophlebitis, a percentage of 1.5; ten of the patients recovered, and five died, a mortality of 33.3 per cent. Dr. Lillie eliminated certain cases in which death occurred from other causes and obtained a mortality of 10 per cent. If I am allowed this privilege my mortality may be brought to about the same figure.

In my series, the infection was on the left in seven and on the right in eight, proving nothing as to the prevalence of sinusitis on either the right or the left.

Various methods of treatment were employed. In eight cases the internal jugular vein was ligated, without cutting, in addition to the operation on the sinus; and in four cases the jugular vein was both ligated and severed, and the facial vein ligated as well. The veins were not resected in the present series. In a number of these cases intravenous medication was employed with such chemicals as metaphen, acriflavine base, mercurochrome and Pregl's solution, as well as with whole or citrated blood. Whole or citrated blood was by far the most effective. The Ayer-Tobey or Queckenstedt test was done in several cases and seemed of doubtful value.

Dr. James B. Costen, St. Louis: One could have no more instructive lesson than this excellent analysis of fifty-two cases of otogenic sepsis by Dr. Lillie. It reminds one that the problem is one of attack and not one of expectant treatment. Examination of cadavers reveals evidence of an occasional healed, untouched thrombosis of the lateral sinus. However, few patients, especially children, can long sustain the daily inroad of sepsis.

For comparison, the results of treatment in the last fifty-two cases of lateral sinus phlebitis at Barnes and St. Louis Children's hospitals were reviewed. Forty

of the patients were children between the ages of 2 and 13, and twelve were adults between 16 and 66. In the same period 1,145 children and 275 adults were treated surgically for acute mastoiditis. The ratio in this group of children to adults having acute mastoiditis is 4:1 and of children to adults acquiring an infection of the blood stream, 3:1. Further, the children having infections of the blood stream represented 3.4 per cent, and the adults, 4.4 per cent, of the total number of patients with acute infections of the mastoid.

The organism almost universally recovered from the wound was Streptococcus haemolyticus, six of the forty children showing various other organisms. Positive blood cultures were obtained from thirteen of forty children, as contrasted with two of twelve adults.

Although children show the loss of ground that goes with the symptoms of invasion of the blood stream, the rest and observation period of a day or so mentioned by Dr. Lillie was carried out in most of our cases before it was decided to open the lateral sinus. Supportive treatment, such as frequent small transfusions of blood, were used early and late, both before and after the operation. Much emphasis was placed on the use of a small transfusion the day before operation.

The question of the mortality rate increasing in cases in which infection of the blood stream follows operations on the mastoid process is raised by Dr. Lillie. Examination of our charts seems to corroborate his assumption that "early diagnosis may be masked by other physical findings." Study of many of these temperature curves show them to reflect a relief from absorption at the time of simple mastoidectomy, but to maintain a miniature septic fluctuation, and assume the marked obvious septic curve days or weeks later. Barring manual injury to the sinus at operation, and perhaps at the time of dressings, I would feel that the phlebitis was usually present in spite of a grossly normal appearing wall of the sinus, and that it spread when the conditions of the wound and blood stream most favored it. I think the care of the wound the first few days after a simple mastoidectomy, when the dura or vein are exposed, has much to do with the chances of infection of a normal wall of the sinus. For some years we have relied on a petrolatum-soaked gauze drain, allowing it to remain until it loosens in the wound from the tissue reaction, usually in from three to five days. Any protective reaction which has thus been stimulated in the dural or venous wall is not disturbed nor is the vein endangered at dressing.

DR. H. I. LILLIE: The incidence of infection may be different in my series because the patients come from outlying districts. The cases which show some of these untoward symptoms are the ones that give the best results. The incidence is about the same as that reported by Dr. Coates.

At the clinic we like to give 500 cc. of blood if necessary, and repeat the transfusion with 250 cc. or less.

Dr. Coates suggested that the mortality rate was low because I eliminated cases. The mortality rate was 13.5 per cent, but one patient died of pneumonia or empyema, which brought the mortality down to 10.6 per cent. . . . I did not eliminate a case in which, on examination, the sigmoid sinus was found to be normal and in which the patient died of intracranial involvement.

In my experience the Ayer-Tobey test was very valuable in certain cases. In a recent case in which there was bilateral infection, there was nothing in the physical findings to suggest which side was involved, but the Ayer-Tobey test was positive and the infection extended from the sigmoid into the bulb.

Dr. Costen's remarks about the sinus wall are well taken. I recall such a case of thrombophlebitis. The impression of the gauze in the wall of the sinus could be seen, and I think this may have been a factor in the trouble in the sinus.

DIAGNOSIS AND TREATMENT OF PRIMARY MALIGNANT NEOPLASMS OF THE MAXILLARY SINUS

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This paper deals with the diagnosis, treatment and end-results in 21 cases of primary malignant tumor of the maxillary sinus. The patients were treated by the department of roentgenology of the Hospital of the University of Pennsylvania. Surgical procedures, when indicated, were performed by the department of otolaryngology of the same hospital. Tumors having their origin outside the antrum were excluded from this series. In a few advanced cases, especially when both the maxillary and the ethmoid sinuses were involved, it was impossible to determine with certainty that the antrum was affected first.

HISTORY

Although it had long been known that tumors of the antrum of Highmore occurred, it was not until 1898, when Phillips [1] presented his paper on this subject, that any comprehensive consideration of this particular lesion was attempted. At this time 16 cases found in the literature and a case observed by the author were reported. Since then the experiences and methods of many workers have been made available. Holmgren [2] reported 18.6 per cent of cures after three years in a series of 59 cases in which total resection of the superior maxilla by diathermy was performed. The operative mortality from this procedure was 5 per cent. These figures do not differentiate primary antral neoplasms from the secondary invaders and tend to raise the percentage of cures, for it is well known that tumors arising in the alveolus and secondarily involving the antrum usually give a higher percentage of cures than primary lesions of the antrum. This is due to the obvious causes of early diagnosis and accessibility for treatment.

From the Department of Otolaryngology of the University of Pennsylvania.

Read before the Section on Laryngology, Otology and Rhinology at the Eighty-Fourth Annual Session of the American Medical Association, Milwaukee, June 16, 1933.

1. Phillips, W. C.: Primary Epithelioma of the Antrum of Highmore, J. Laryng., Rhin. & Otol. **13**:325, 1898.

2. Holmgren, Gunnar: Erfahrungen über chirurgische Behandlung von malignen Oberkiefertumoren, Acta oto.-laryng. **7**:511, 1925.

In the 84 cases reported by Greene [3] 69 patients received treatment. Of 36 patients treated by operation and irradiation, 33.3 per cent were living after from one to five years and 16.7 had passed the three year limit at the time the article was published. Thirty-three of the patients were treated by radium alone. Of these only 1 was well two and one-half years later. Greene, therefore, advocated wide open operation followed by irradiation.

Barnes,[4] too, on account of his experiences with 25 cases, expressed the belief that operation followed by immediate irradiation holds the most promise of cure. Sixteen per cent of the patients so treated had passed the four year period.

The report of New [5] embraces a total of 186 patients observed. Of these, 129 had primary lesions of the antrum. Seventy patients received treatment. Thirty per cent had passed the one year period, and 22.8 per cent were living after three or more years. New stressed the value of Broder's grading of neoplasms in estimating the prognosis and type of treatment, advocating cautery or diathermy for tumors of the lower grades and relying on surgical intervention and immediate irradiation for the higher grade epitheliomas and sarcomas.

Sharp [6] stated that at Memorial Hospital in New York a special technic of irradiation which measures exactly the dose of radiation given has been developed. Operation is used only to provide exposure and drainage. Sharp found aspiration biopsy, following the technic of Martin and Ellis, to be satisfactory, and he advocated its use as a routine procedure in all diseases of the antrum.

Quick,[7] in 1930, offered some interesting conclusions: 1. Surgical exploration and biopsy should be done earlier for early diagnosis. 2. Surgical principles in treating new growth elsewhere do not apply in the sinuses, with few exceptions. 3. Radium and roentgen radiation are of value and, except in palliative procedures, should be used in conjunction with surgical treatment. 4. Radium and roentgen radiation, if used in sufficient dosage, can be depended on to eradicate the lesion. 5. Surgical intervention must be used to secure exposure and drainage. 6. The anatomy of the parts makes infection more of a hazard than elsewhere.

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3. Greene, D. Crosby: Carcinoma of the Antrum, Am. J. Roentgenol. **9**:591, 1922.

4. Barnes, H. A.: Malignant Tumors of the Nasal Sinuses, Arch. Otolaryng. **6**:123 (Aug.) 1927.

5. New, Gordon B.: Malignant Tumors of the Antrum of Highmore, Arch. Otolaryng. **4**:201 (Sept.) 1926.

6. Sharp, G. S.: Carcinoma of the Maxillary Antrum, Radiology **17**:984, 1931.

7. Quick, D.: Treatment of Malignant Growths of the Nasal Accessory Sinuses and Nasopharynx, Radiology **14**:191, 1930.

DIAGNOSIS

One might say that the prognosis varies inversely with the ease of diagnosis. The advanced, easily recognized lesion offers little hope of cure, whereas the early growth, which unfortunately often is symptomless, can frequently be eradicated if proper measures are followed. The surgeon need not feel chagrined if in his honest effort to diagnose this type of lesion he opens some maxillary sinuses and finds no neoplasm. The surgical exposure and inspection of the interior of the maxillary sinus can be carried out under local anesthesia with a relatively slight element of risk.

The symptoms of early lesions are practically nil. The tumor must be fairly large before it gives rise to a sense of fulness in the antrum and must erode the bony wall of the sinus before tumefaction of the soft tissues of the cheek occurs. With growth, however, the initial symptom is pain. Of necessity, some of the nerve filaments of the antral mucosa are involved, this in turn giving rise to localized or radiating pain varying from slight discomfort or parethesia to agony. Too often this type of symptom is given the meaningless diagnosis of neuralgia and the patient is permitted to drift. One patient in this series was operated on for tic douloureux before the correct diagnosis was made. In the early stages transillumination, antral puncture and lavage and intranasal examination will show nothing. Roentgen examination, with and without filling of the sinus with an opaque oil, will frequently do more than any other method to suggest the presence of a growth or at least arouse a suspicion of such a lesion. In advanced tumors, in which obvious abnormality of the antrum exists, the diagnosis is made without difficulty, provided the surgeon is sufficiently daring to explore the antrum and remove biopsy specimens. I have had no experience with aspiration biopsy.

The patient who presents himself on account of facial pain or discomfort should have thorough study. Syphilis, dental disease, antral infections, ocular disorders, trifacial neuralgia and neuralgia of the sphenopalatine ganglion should all be excluded by the various methods of diagnosis available. In the event that no satisfactory explanation for the pain is discovered and the roentgenogram is suggestive of antral disease or neoplasm, exploratory antrotomy should follow. In spite of these statements, however, one should not entertain the idea that this type of lesion is common. On the other hand, it is relatively rare. Ewing [8] reported that of 1,892 cases of malignant growths only 2.53 per cent were of nasal origin. The exact proportion of antral lesions is not stated, but one can safely assume that it would be a fairly small fragment of this 2.53 per cent.

8. Ewing, cited by Greene.[3]

TREATMENT

An absolute diagnosis cannot be obtained without the securing of a biopsy specimen, which of course necessitates the opening of the antrum. The method of choice is to perform a Caldwell-Luc operation under local anesthesia in cases in which the findings are suggestive of an antral tumor. This permits inspection of the sinus and the securing of biopsy material. In a few cases in which the growth filled the antrum the specimen was secured through an intranasal antral window

TABLE 1.—*Types of Antral Neoplasms*

	Patients
Carcinoma	19
Sarcoma	2
	21

TABLE 2.—*Sex of Patients*

	Patients
Males	11
Females	10
	21

TABLE 3.—*Ages of Patients*

Years	Patients
1–10	0
11–20	1
21–30	0
31–40	3
41–50	2
51–60	9
61–70	5
71–80	0
81–90	1
	21

via the inferior meatus. Sometimes the gross appearance is so typical of a neoplasm that radium is at once applied before an absolutely positive pathologic diagnosis is secured.

If the interior of the sinus is negative for tumor, immediate closure is performed. In case the interior of the sinus has a suggestive but not definitely positive appearance, the wound is packed wide open.

When the positive diagnosis is obtained, the patient receives roentgen therapy over the antrum and the cervical lymphatic channels as a preliminary procedure. In this series the dosage was carried to the limit of cutaneous tolerance with high or low voltage therapy. The doses given to patients treated with high voltage therapy varied from 800 to 1,600 roentgens per portal. The factors were: 200 kilovolts, a

distance of 50 cm., 4 milliamperes and a 0.5 mm. filter copper. Some of the patients received in addition an erythema dose of radium delivered through a 3 cm. radium pack, the filter being equivalent to 3 mm. of lead. After operation radium is placed in the maxillary sinus; the dosage in this series ranged from 300 to 4,000 milligram hours.

TABLE 4.—*Duration of Symptoms Before Examination*

Duration	Cases
2- 4 months	4
4- 6 months	2
6-12 months	6
12-18 months	1
18-24 months	2
2- 3 years	3
Onset of symptoms not known	3
	21

TABLE 5.—*End-Results of Treatment*

	Patients
Dead	17
Living after more than 18 months	4
	21

TABLE 6.—*End-Results in Fifteen Surgical Cases*

	Patients, per Cent
Living after more than 1½ years	26.6
Living after more than 5 years	13.3

TABLE 7.—*Analysis of Cases of Four Living Patients* *

Case	Years Living Since Treatment	Time Between Symptoms and Treatment, Months	Lesion
1	13	7	Sarcoma
2	5	8	Carcinoma
3	2	10	Carcinoma
4	1½	4	Carcinoma
Percentage of patients living more than 1 year			19
Percentage of patients living more than 5 years			9.5

* Death occurred in all cases in which symptoms had been present more than ten months before treatment was started.

In the 21 cases here reported, there were 19 carcinomas and 2 sarcomas. One sarcoma occurred in a child, and the other in an adult. The age incidence in general shows that more than half of the cases occurred in persons between 50 and 70 years of age. Four of these patients, or 19 per cent, are now living after one and one-half years or longer. One patient has been living for thirteen years without signs of recurrence; another, five; another, two, and still another, one and

one-half. Only 9.5 per cent, therefore, have passed a five year period. It is interesting to note that in no case in which symptoms had existed for more than ten months prior to the beginning of treatment was a cure effected.

One interesting characteristic of all these cases was the infrequency of metastasis to the neck and chest. Spread, when it occurred, was by contiguity, extending upward and backward and involving the adjacent sinuses, the orbit and sometimes the cranial cavity. In many cases, too, anterior and lateral extension took place, the process breaking through the bony wall and invading the tissues of the cheek.

The treatment followed depended largely on the status of the lesion. Six growths when first seen were so far advanced as to be considered inoperable. All the patients, however, regardless of the condition existing when they were first seen, were treated with roentgen irradiation and radium. When pain was marked, avulsion of the sensory root of the trigeminal nerve was performed or, if this operation was not considered proper, alcohol was injected into the second and third divisions. Radical removal of the upper jaw was performed in 3 cases, in all of which the patients had a recurrence and died within a year. In 10 cases radical operation was performed on the maxillary sinus involved. Three of the patients operated on received electrocoagulation of the lesion at the time of operation.

Of the 21 patients treated, 4 are alive (19 per cent of the entire series and 26.6 per cent of the 15 operable patients). Two of these (13.3 per cent of those operated on) have passed the five year period.

CONCLUSIONS

1. Early diagnosis of antral neoplasms is necessary for the best therapeutic results.

2. If an antral neoplasm is suspected, the sinus should be opened for inspection and biopsy.

3. Surgical procedures should have as their major purpose exposure of the lesion, so that direct irradiation may be used.

4. Surgical procedures are necessary for the maintenance of adequate drainage.

5. Roentgen and radium therapy in adequate dosage offer the most hope of cure.

Dr. H. K. Pancoast and Dr. E. P. Pendergrass made available the case histories of the patients mentioned in this paper.

ABSTRACT OF DISCUSSION

Dr. Edward C. Sewall, San Francisco: It seems that roentgen examination is the most important means available for early diagnosis. Signs of malignant

growths of the antrum recognizable by other methods present themselves comparatively late. Pain and tumefaction appear often when the lesion has passed beyond the stage when eradication is possible. Roentgen examination will, however, often distinguish even slight changes in the thickness of the lining mucosa of the antrum. This is best recognized by means of the stereoscopic technic. Opaque oils may at times be employed, but ordinarily they are not necessary for the recognition of changes in the mucous membrane. My associates and I employ stereoscopic study of the sinuses in practically all our rhinologic cases. It seems important to have the fundamental facts gained thereby even when a personal sacrifice in compensation is entailed.

It is not necessary to open a normal antrum for diagnostic purposes. It is rather a question of inspecting to distinguish the nature of the pathologic process that is shown frequently by the x-ray films.

Certain difficulties present themselves in the use of roentgen examination for diagnostic purposes. After an antrum has been operated on the difficulty of interpretation is much increased. Definite evidence of bony involvement does not always signify malignancy.

Dr. M. R. Guttman, Chicago: I wish to add a few observations derived from the study of 69 patients with primary carcinoma of the antrum seen by my associate, Dr. Joseph C. Beck, and me.

The patients are divided into two groups, those seen prior to 1924, of whom there were 42, and those seen since 1924, of whom there were 27. The first group were operated on by radical resection of the jaw with the cold knife or by means of the cautery method described by Dr. Gordon B. New. Only 1 of the 42 patients showed no recurrence. This man had a radical resection of the upper jaw, and died of pneumonia fifteen years after operation. Of the 27 patients seen since 1924, 5 have shown no recurrence, 1 after seven years, 1 after five years, 1 after three years and 1 after two years. These 57 patients were treated by a combination of irradiation and electrothermic operation. In all of the patients that showed no recurrence the malignant growth was in the antero-inferior portion of the antrum. Furthermore, the growth was an adult, fully differentiated type of squamous cell carcinoma and was not associated with palpable glands of the neck. The prognosis appears to depend on the location of the neoplasm in the antrum, growths lying anterior and inferior to a plane passing through the canthi of the orbit and the angle of the jaw being most favorable. From its biologic characteristics the adult, fully differentiated neoplasm is more amenable to treatment, while the embryonic, anaplastic types are more or less hopeless. The presence of metastases in the neck makes for a gloomy prognosis.

The results of therapy, in our series at least, are not brilliant, but 6 patients in a series of 69 showing no recurrence. Apparently, in our hands irradiation combined with an electrothermic procedure has produced much better results than the cold knife or actual cautery as practiced by New.

Dr. Fred Z. Havens, Rochester, Minn.: In the treatment of malignant disease of the maxillary sinus the use of cautery (either actual or electrosurgical) combined with irradiation offers a better prospect of permanent benefit than any other method yet proposed.

Dr. Houser referred to a report by New, in 1926, on the use of this method in the treatment of 70 patients having primary malignant tumors of the antrum. A recent survey of the results obtained in that series revealed that 12 (17.1 per cent) of the 70 patients were alive and well nine or more years after treatment (table 8).

Teamwork of a special type is necessary to obtain the best results in the treatment of these patients. There must be a pathologist who has had wide experience with tumors occurring in this region. It is not enough for him to say that a given tumor is malignant; he must have a working classification so that the surgeon may be informed as to the type of tumor with which he is dealing. The plan of treatment will vary materially, depending on whether the tumor is high grade or low grade. A tumor of the antrum which is classified as high grade may be cured by means of irradiation, whereas one of low grade can be treated much more effectively by cautery.

TABLE 8.—*Twelve Nine Year Cures Among Seventy Patients*

Year Treated	Age, Years	Sex	Diagnosis	Period Without Recurrence (to April, 1933)
1917	19	M	Fibrosarcoma	15 years, 4 months
1918	17	M	Fibrosarcoma	14 years, 10 months
1918	63	F	Sarcoma	14 years, 9 months
1918	57	M	Squamous cell epithelioma, graded 3	14 years, 7 months
1919	19	F	Lymphosarcoma	13 years, 7 months
1920	70	F	Lymphosarcoma	13 years
1920	40	M	Squamous cell epithelioma, graded 2	12 years, 8 months
1920	20	M	Mixed tumor	12 years, 4 months
1920	25	F	Squamous cell epithelioma, graded 3	12 years, 7 months
1921	41	M	Squamous cell epithelioma, graded 4	11 years, 11 months
1922	45	F	Squamous cell epithelioma, graded 2	11 years, 1 month
1924	72	F	Squamous cell epithelioma, graded 3	9 years

The surgeon must be thoroughly familiar with the problems peculiar to operation in this field. He must understand the behavior of the various types of malignant lesions in order to know whether cautery alone, radium alone or some combination of the two will give the best results.

I endorse Dr. Houser's point that exploration should be done when the question of malignancy cannot be settled otherwise, and that one need not feel chagrined if exploration gives negative data.

Quick pointed out that infection, with the resulting inflammatory process, complicates diagnosis, adds to surgical risk and aggravates tumor growth. When exploration is done and later the patient is referred to another surgeon for treatment, the inflammatory reaction resulting from the exploration may make it difficult or impossible to ascertain the exact nature of the growth or to determine its limits accurately, and the patient thus may lose whatever chance he may have had of being cured.

Clinical Notes

TREATMENT OF HAY FEVER BY NASAL INJECTIONS OF ALCOHOL

HARRIS H. VAIL, M.D., CINCINNATI

One has only to read the book by Coca, Walzer and Thommen[1] to realize what an enormous problem hay fever is. Possibly at the present time the cycle of 1881 is being repeated in which, according to these authors, "it seems that every conceivable method of rhinologic procedure had been thought of and enthusiastically advocated." These procedures were galvanocautery, correction of intranasal abnormalities, massage of the nasal mucosa, resection of the ethmoid sinus, intranasal application of hot air, operative procedures on the antrum, excision of the tubercle of the septum, resection of the nasal nerve and injection of alcohol into the nasal nerve.

As the specialty of allergy has developed, that of rhinology has had less to do with hay fever. The patients with hay fever seen today by the otolaryngologist are those who have been unrelieved by injections of pollen, or whose condition has been hitherto undiagnosed or who are unable, for various reasons, to seek more favorable localities after the onset of the hay fever.

Therefore, it seems to me that a preliminary report on a small series of patients treated with injections of alcohol into the nose might be of interest. In 1931, the result of injections of alcohol given by one of my colleagues, Dr. Edward King of Cincinnati, into the nose of my office nurse aroused my attention. This young woman had had the autumnal type of hay fever for four or five years, with increasing severity in her symptoms each year. Antral lavage and the usual rhinologic treatment gave only temporary relief. The injections of pollen caused such severe reactions that they had to be abandoned. Dr. King gave her the injections of alcohol into the nose at the beginning of the hay fever. I must confess that I was a bit skeptical about the outcome but, greatly to my surprise, throughout the season of 1931 she experienced marked relief. I examined her nose frequently, and at all times during the hay fever season the mucosa of the turbinates and septum was a healthy pink. Through the winter of 1931 her nose behaved as any normal nose would have done. I was convinced of the value of the treatment and its lack of undesirable after-effect in this particular case.

On looking up the literature on the subject, I found an article by Leichsenring,[2] and in 1932 I began to use his technic. Briefly, this is to inject 1 or 2 cc. of from 40 to 60 per cent alcohol with procaine hydrochloride into the head of

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Read before the Section on Laryngology, Otology and Rhinology at the Eighty-Fourth Annual Session of the American Medical Association, Milwaukee, June 16, 1933.

1. Coca, A. F.; Walzer, M., and Thommen, A. A.: Asthma and Hay Fever in Theory and Practice, Springfield, Ill., Charles C. Thomas, Publisher, 1931.

2. Leichsenring: Management of Allergic Diseases with Injection of Alcohol Into the Nasal Mucosa, Ztschr. f. Hals-, Nasen- u. Ohrenh. **28**:220 (May) 1931.

the lower turbinate and septum opposite it, after cocainization of the site of injection, rarely to make an injection into the middle turbinate and by all means to avoid the olfactory region. Leichsenring stated that the injection caused no noteworthy symptoms—mostly a local burning and twinges of pain to the antrum and teeth. He stated that the latter symptoms were all over in two days. He reported good results in 80 per cent of the cases and advised that the injections be made yearly.

I wish to report the results in twelve persons treated by me. Three had so-called "rose fever" and obtained complete relief from their symptoms. The remaining nine patients had autumnal hay fever. Five of them obtained marked relief. One of these patients had the spring, summer and fall type of hay fever. He was not given an injection until August, 1932, and it was necessary to make two injections, the second one being into the middle turbinates, before he obtained complete relief. Another patient was a boy of 10 with severe asthma and hay fever. Complete relief from the hay fever was obtained, but the asthma was just as severe as before treatment. He had to go north for the remainder of the season for relief from the asthma. Four patients had no relief from their symptoms.

In the autumnal form of hay fever my results were a little better than 50 per cent. In the spring form, my results in three cases were 100 per cent.

The injection of alcohol into the turbinates and septum is not as painless as Leichsenring stated. The patient experiences severe shooting pain through the temple, cheeks, nose and teeth for ten or fifteen minutes. This gradually clears up in the course of twenty-four hours. Bleeding, though never severe, is rather annoying and lasts for the same length of time. As soon as the injection is made there is noticed an extreme blanching of the mucous membrane extending over the lower turbinate into the middle meatus as well as on the septum to a corresponding level. The septal injection is more difficult to make. The nasal mucosa of the relieved patients after twenty-four hours has a normal pink color, and the patient goes through the hay fever season free from nasal symptoms. Some of the patients showed marked improvement in lacrimation. The mucosa of the unrelieved patients appeared just as it did before injection. The one patient in whom a second injection was made showed pink, normal inferior turbinates with pale, swollen middle turbinates until the injection was completed, after which the middle turbinates assumed a normal color.

So much for this kind of injection of alcohol into the nose for hay fever. It seems to me, however, quite apropos to speak of another form of injection which is being advocated and which I feel impelled to criticize severely; that is, the injection of alcohol into the sphenopalatine ganglion for hay fever and vasomotor rhinitis. Ruskin [3] stated that in some cases of Meckel's ganglion he observed congestion of the nose of from a few days to two or three months' duration which was severe enough to cause discomfort. He advocated this form of treatment in cases of atrophic rhinitis not caused by sinusitis. He reported some cases of hay fever in which injections made in the same manner brought relief.

Walsh [4] reported the results of injections of alcohol into the sphenopalatine ganglion in cases of vasomotor rhinitis completely divorced from protein sensi-

3. Ruskin, S. L.: Atrophic and Vasomotor Rhinitis, Arch. Otolaryng. **11:** 689 (June) 1930.

4. Walsh, T. E.: Vasomotor Rhinitis: Its Treatment by the Injection of Alcohol into the Sphenopalatine Ganglion, Arch. Otolaryng. **16:**83 (July) 1932.

tivity, bacterial allergy, endocrine dysfunction, neurosis and anatomic nasal abnormalities as etiologic and predisposing causes. He stated that he used Ruskin's technic with slight modifications. A study of his article shows that there is a little inconsistency with the object of the paper. Namely, in the ninety cases reported, skin tests were done in thirty-nine with positive results in twenty-seven, so that some of the cases undoubtedly were allergic. Of the ninety cases, failures were obtained in only eight. Walsh spoke of the severe pain in the region of the temple following the injection, with headaches for one or two hours followed by swelling of the face, usually localized to the cheeks, lasting from two days to a week in the patients who were relieved. Those who were unrelieved had no swelling. As the swelling subsided, the nasal condition improved. He stated that there was a tendency for any polypi present to atrophy.

I cannot understand why a blind injection of alcohol into Meckel's ganglion in cases of neuralgia of Meckel's ganglion will cause vasodilation of the nasal mucosa, while the same injection in cases of vasomotor rhinitis will cause vasoconstriction of the mucosa.

Vogel,[5] in 1929, stated that the present knowledge of the function of the sphenopalatine ganglion and of the symptoms of its disease shows that the understanding of these subjects is based merely on conjectures. He examined the sphenopalatine ganglion in a patient with atrophic rhinitis who died of heart trouble, and found on microscopic sections definite pathologic changes in the sphenopalatine ganglion. The gasserian, ciliary, geniculate and jugular ganglions were normal. The changes in the sphenopalatine ganglion were a marked development of connective tissue between the walls of the ganglion, a disappearance of these cells and atrophy of the ganglion with round cell infiltration in the region of the small veins of the perigangliónic space which became more frequent and abundant toward the nasal mucosa. In addition, the ganglion and its nerve stems showed round lymphocytic and leukocytic cell infiltration along the vessels quite similar to that seen in the nasal mucosa.

To find out more about the relation between ozena and the changes in the sphenopalatine ganglion, Vogel removed one ganglion in ten patients on whom he was performing the Lautenschlager-Seiffert operation for ozena. He found in all ten ganglions the same pathologic changes.

It is important to note the after-results of the extirpation of the sphenopalatine ganglion. Vogel stated there was never any noticeable after-result. There was slight anesthesia about the face and in the nose lasting for about a month. There was no difference in the color of the nasal mucosa or its secretion. Tear secretion was not interfered with. There was no increase or decrease of the atrophic rhinitis after extirpation of the ganglion in a person with healed tertiary syphilis who showed no ozena. Vogel's conclusions were: 1. In genuine atrophic rhinitis there is atrophy of the sphenopalatine ganglion. 2. There can be found no proof that any primary disease of the ganglion is the cause of the atrophic rhinitis. The disease of the ganglion must be regarded as secondary, and for the development of ozena, some other cause, up to now unknown, must be considered. 3. It is likely that the function of the ganglion can be taken over by other nerves.

When no change has resulted in the nose after the surgical removal of the sphenopalatine ganglion, in man as well as in animals, how is one going to account

5. Vogel, Klaus: Histopathologische Befunde am Ganglion Sphenopalatinum mit besondere Berücksichtigung der atrophischen Rhinitiden, Ztschr. f. Hals-, Nasen- u. Ohrenh. **22**:507, 1929.

for all the changes that are reported by Ruskin and Walsh following the injections of alcohol into the sphenopalatine ganglion? I am inclined to look with a little suspicion on one method which causes two opposite results, namely, a shrinkage of the nose when one wants to shrink it, and a dilation when one wants that.

I have performed a good many dissections of the sphenopalatine ganglion, and I should not want to have alcohol injected into my ganglion. I have, as a routine measure in the past, injected procaine hydrochloride into the region of the ganglion for nerve block in nasal operations, and I was always thankful when the patient left the hospital without any complication in the sphenomaxillary fossa. One has only to dissect a ganglion or to examine a microscopic slide to realize how vascular the sphenomaxillary fossa is and that any injection which has to be done blindly gives no real surety that there will not be a puncture of a vessel in the fossa with a resulting infection. There can be found in the literature reports of cases of severe complications and even fatalities following the injection of procaine hydrochloride into the sphenomaxillary fossa, so one should not take a chance of complicating a case of hay fever or atrophic rhinitis by an abscess in the sphenomaxillary fossa or paralysis of one of the cranial nerves.

In a rather general way, the after-effects of alcohol injected into the nerve structure are known by all. May [6] concluded after his experiments that alcohol injected into the trunk of peripheral nerves produces a more or less complete local necrosis of the nerve at the point of injection. The change is not an ascending one, the nerve above the injection being normal. The cells of origin of the fibers may show some degree of chromatolysis but do not exhibit signs of permanent injury. The conditions produced by an injection of alcohol are more favorable to regeneration than those resulting from simple section without suture. The anatomic continuity of the nerve trunk favors rapid regeneration, though this is to some extent retarded by the fibrosis which occurs to a greater or a lesser extent in every case in which injection of alcohol is done. This last conclusion is one to be kept in the back of one's head if one is using injections of alcohol into nerve structure. In other words, according to May, not only will there be a regeneration of the nerve but there will also be some scar tissue. When one is dealing with such a painful condition as neuralgia of the trifacial nerve, I am sure injections of alcohol into the gasserian ganglion are justified, but the same cannot be said when one is dealing with a relatively minor condition like hay fever.

Injection of alcohol into the inferior turbinates and septum affects the terminal nerves, hence its action is temporary and probably confined to the network of nerves about the cavernous spaces of the turbinate, causing a temporary paralysis of the vasodilators and vasoconstrictors. The latter must be also affected, as there is none of the shrinkage of the turbinate such as follows the use of epinephrine. As I have said before, the color of the mucosa remains a healthy pink.

In conclusion, I think that the injection of from 40 to 60 per cent alcohol with procaine hydrochloride into the inferior turbinates and beneath the mucosa of the nasal septum, and in some cases into the middle turbinates, is worth trying in cases of so-called hay fever in which pollen therapy has been unsuccessful and in which the patient is unable to seek a more suitable climate. The effect of a successful injection of alcohol lasts from six to eight weeks. In that period of time the nasal mucosa of the area into which injection is made preserves a normal pink color, and the size of the turbinate is within normal limits. The only drawback to the injection is the severe pain noticed at the time which lasts about a day. Other than that

6. May, Otto: The Functional and Histological Effects of Intraneural and Intraganglionic Injections of Alcohol, Brit. M. J. **2**:465 (Aug. 31) 1912.

no ill effects have been noted. After twenty-four hours there seems to be no sensory disturbances of the mucosa. A second injection may succeed when the first injection has failed.

ABSTRACT OF DISCUSSION

Dr. Frank J. Novak, Jr., Chicago: I shall limit my discussion to that portion of the paper which deals with the injection of alcohol into the inferior turbinate and the septum. Dr. Vail made it clear that he does not offer the procedure as a solution of the problem of hay fever. I do not believe that any further development, modification or refinement of the desensitization technic offers any hope of the solution of this problem.

When alcohol or water is injected into an area of the nasal mucosa the progress of the diffusion of the fluid may be observed by ultraviolet illumination to the exclusion of all other light. The area into which injection is made promptly assumes the shape, with irregularities corresponding to the pseudopods, which one observes in the cutaneous reaction following the injection of an allergen in a sensitive subject. It seems to me that an area mapped out in this manner, especially if it shows a urticarial response, however slight, is the area to be studied cytologically. If there is a cessation of any symptoms of hay fever in the patient, one may logically ask the following questions: 1. Has there been a gross mechanical disruption of the minute nerve fibers in this area? 2. Is the effect due to the chemical action of the alcohol on the nerves? 3. What are the properties of the cells of this area, particularly as far as permeability and irritability are concerned? Do these cells differ from those of a normal person as to permeability and irritability?

Dr. Vail's paper is important because it contains suggestions for a new line of attack on the problem of hay fever, an investigation of certain crucial areas of the nasal mucosa which may be located clinically by injection of alcohol and studied by the newer cytologic technic.

I agree completely with everything Dr. Vail has said in regard to injections into the sphenopalatine ganglion.

Dr. Virgil J. Schwartz, Minneapolis: Hay fever is but one small part of the various groups of sensitization phenomena.

1. Anaphylaxis is an acquired sensitization, never inherited, due to a soluble foreign protein (the antigen) which produces antibodies in the blood of the animal into which injection has been made, this production reaching its maximum in from seven to ten or more days. Then, if the antigen is again injected, it combines specifically with the antibodies, causing a reaction or shock.

2. Allergy is a type of sensitization in which an organism develops a changed reaction capacity through recovery from a bacterial disease (e.g., tuberculin reaction) or through treatment with foreign substances such as serums, drugs and foods. Allergy may be acquired or inherited.

3. Atopy includes only conditions of human hypersensitiveness which are practically always inherited, not acquired. In this class come hay fever and asthma.

In allergy or atopy, a diet or a drug such as hydrochloric acid, tending to shift the hydrogen ion concentration of the blood toward the acid side, may greatly relieve the allergic symptoms. In anaphylaxis, however, a shift to the alkaline side reduces the tendency to anaphylactic shock or reaction. In allergy there is an abundance of eosinophils in the secretions, the nasal mucosa and the blood. Two basic physiologic changes may be present: increased smooth muscle irritability, as in asthma, or increased capillary permeability, as in vasomotor rhinitis, or both.

Wassermann injected alcohol into the ethmoid and nasopalatine nerves in twenty-nine cases of hay fever, with good results. Wassermann's article prompted Leichsenring to report the results in his series of cases. Wagner had suggested extirpation of the nasal nerves for relief from hay fever, but this being too difficult, Leichsenring tried blocking the nerves by injecting alcohol into the middle turbinate. Later he made injections into the inferior instead of the middle turbinate, a less disturbing procedure, with 80 per cent good results.

Sluder made injections into the sphenopalatine ganglion through the posterior tip of the middle turbinate. Walsh reported excellent results after injections into the ganglion, but the immediate secondary effects (pain and swelling) were disagreeable. Vail's procedure has the advantage that the symptoms incident to the injection are over in twenty-four hours, so that even if there is no marked improvement in the hay fever, at least the patient has not experienced much discomfort.

There is food for thought in Dr. Vail's criticism of Ruskin's claim that he can produce vasodilation in atrophic rhinitis and vasoconstriction in vasomotor rhinitis by the same procedure, namely, blocking the sphenopalatine ganglion with alcohol. Perhaps both dilator and constrictor fibers are blocked at once, and that since whichever had been overacting previously is now obtunded, symptomatic relief appears.

Dr. M. F. Arbuckle, St. Louis: It is my belief that the chief etiologic factor in hay fever may be referred to as allergic. The symptoms apparently are the result of a disturbance of the involuntary nervous system which, in turn, may be the reason why one has so much difficulty in treating these patients, since the lack of understanding of this system is well recognized. Vasomotor rhinitis is the result of some allergic disturbance as expressed through impulses transmitted by way of the vegetative nervous system.

I wish to call attention to the communications of Greenfield Sluder concerning the anatomy of the nasal ganglion and his experiences with the use of cocaine in the control of symptoms transmitted through this nerve supply and concerning the application of cocaine to the nasal ganglion and the nasociliary nerve in securing anesthesia for operation on the lateral wall of the nose.

I have had no experience with injection into the nasal ganglion in the treatment of hay fever or other allergic disorders. This procedure is attended by considerable danger and should be attempted only by those well acquainted with the anatomic structures involved. Serious hemorrhage and damage to the optic nerve are complications which I have observed.

A better understanding of allergy will accomplish better results in the treatment of nasal disorders, such as hay fever. This will be accomplished only when it is acknowledged that allergy is a specialty within itself, for the most successful practice of which special training is required.

I congratulate Dr. Vail on his success with the treatment of hay fever by the injection of alcohol beneath the nasal mucous membrane. I wonder if the reason for cessation of symptoms after the injection of alcohol might be the same as obtains, for example, after a submucous resection or other operation on the nose, in other words, a temporary loss of function due to trauma.

Dr. Harris H. Vail, Cincinnati: Rhinologists are beginning to see a number of patients who cannot go through all the tests and pollen therapy and who are not able to go away for the hay fever season. The injection of alcohol is a practical method that gives at least 50 per cent good results, and with improvement of technic will do even better.

I was asked to give the dosage of alcohol. I insert a needle about 1½ inches long into the anterior half of the lower turbinate for about 1 inch, withdrawing it slowly as I inject 2 cc. of alcohol and procaine hydrochloride. The septal injection may be difficult. I usually inject about 0.5 cc. into the septum. As Dr. Novak said, with the ultraviolet rays one can see the material entering the middle meatus. The alcohol causes an intense blanching of the inferior turbinate and middle meatus. I inject 4 cc. of 40 per cent alcohol with 1 cc. of 2 per cent procaine hydrochloride without epinephrine, administering 2 cc. on each side.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology During the Years 1932 and 1933

ADVANCES IN THE FIELD OF ALLERGY AS RELATED TO OTOLARYNGOLOGY

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The literature of the past year on allergy, while interesting, has presented no material radically different from that found in articles published in previous years. It illustrates rather, the development of ideas already advanced, especially along the line of physical allergy in the European countries. A book, "Les maladies de lumierè et leur traitement," by Jausion and Pages, is the most complete treatise on diseases caused by light which has appeared up to the present. I reported light as a cause of urticaria and nasal allergy many years ago but had no idea of the clinical importance of this form of allergy until I made a routine search for it especially in patients whom I had previously thought were sensitive to pollen or foods but who were not relieved therapeutically. I find now that it is a relatively common cause of diseases of the eyes, the eyelids and the skin of the face and hands, as well as a cause of stoppage of the nasal passages and of general illness. In one patient, a baseball enthusiast, nasal allergy was a constant symptom during the baseball season. Stoppage of the nasal passages could be brought about within a moment by exposing the patient's eyes to the light of a large nitrogen lamp. Patients who are sensitive to light are rather frequently sensitive to light and heat combined. These factors should be taken much more seriously by otolaryngologists, for relief can be obtained both by treating the skin generally with the agent responsible for the illness and by protecting the skin and eyes from light by the use of green or black hats and clothing and by the use of glasses which are impervious to the rays of the blue and red ends of the spectrum.

CAUSES OF ALLERGY

The origin of sensitiveness to a particular agent has attracted the attention of students of allergy for many years. The views vary widely. Coca believes that people inherit sensitiveness to some particular agent; other observers believe that exposure to a given agent is an additional

deciding factor. Numerous intermediate views might be pointed out, such as those in which the factor is thought to be exposure to a given agent during an illness of some sort or after anesthesia or a nasal operation. It is not uncommon for symptoms of sensitiveness to certain agents to follow some striking circumstance in a person's life; thus, sensitiveness to heat may appear after a severe burn, or sensitiveness to light after a severe sunburn. One might always bring up the fact, however, that the burn or the sunburn caused by heat or light may have been due to the fact that the patient had been hypersensitive to the agent before the burn occurred and that the sensitiveness was only at that time brought to his attention. This is frequently the case.

Black [1] cited an interesting case in which exposure was followed by the onset of sensitiveness of a given type. An operator in a beauty parlor came to him for asthma caused by orris root, which she had been using in her work. He found all the skin tests negative except that for orris. The patient substituted buckwheat for orris and was free from symptoms for ten months. She then returned with asthma, and, on being tested, was found to be sensitive to buckwheat. Rye flour was substituted for buckwheat. She was immediately relieved but returned at a later date with symptoms caused by rye flour. She was again relieved by the substitution of barley flour for rye flour. At this time she gave positive skin tests for orris, buckwheat and rye but negative tests for other agents which are commonly responsible for asthma.

Sulzberger and Kerr [2] reviewed the subject of sensitization of the eczematous type and reported 10 selected cases illustrating the use of the patch test in diagnosis. They stated that the agents to which a person can become sensitive are almost innumerable. However, they mentioned the fact that there are certain agents which seem prone to sensitize and cause a reaction, such as Rhus toxicodendron and primrose, agents which are frequently used in the treatment of dermatologic lesions and the anesthetics. They mentioned ammoniated mercury, butesin picrate, trinitrophenol, quinine, resorcin, corrosive mercuric chloride and also the local and general anesthetics, including nupercaine. Their observations in this line are in perfect harmony with a remark of Loudermilk: "It seems strange how frequently patients become sensitive to drugs used in the symptomatic treatment of hay fever and asthma." I believe that Sulzberger and Kerr's conclusions in regard to allergic agents can be applied to sedatives in general, including such

1. Black, J. H.: The Development of Sensitiveness in an Allergic Person, J. Allergy **4**:24 (Nov.) 1932.

2. Sulzberger, M. B., and Kerr, P.: Sensitizations of Eczematous Type; Ten Selected Cases Illustrating Some Uses of the Patch Test, J. Allergy **4**:326 (May) 1933.

mild sedatives as acetylsalicylic acid and alcohol encountered in such mild drinks as beer. These investigators emphasized the usefulness of the delayed reaction in patch tests in diagnosis and made the apt statement that drugs or other materials which produce positive skin tests and cause dermatitis by contact might cause generalized dermatitis if they are taken by mouth in adequate dosage. They also emphasized the importance of making patch tests on areas of skin which are inclined to react on natural contact before a given agent is excluded as a cause of illness.

Abramowitz and Noun [3] gave a historic report concerning chloral hydrate as a cause of a variety of lesions of the skin. They added a type of allergy which is scarcely known, contact eczema caused by high grade sensitiveness to chloral. In the patient, a druggist, eczematous dermatitis of the hands, forearms and face were caused by exposure to almost infinitesimal amounts of this drug. Patch tests with a 10 per cent solution of chloral hydrate gave a strong positive local reaction.

Gordon and Lieberman [4] reported idiosyncrasy to viosterol in a rather large proportion of infants who were taking this preparation. The symptoms were diarrhea, vomiting, loss of appetite, colic and loss of weight. This form of allergy has been observed and reported rather frequently. I myself know of several cases, one in a patient who as a baby was given cod liver oil for several years without ill effect and who, several years later, on taking a normal dose of viosterol, had abdominal pain, nausea and vomiting which were sufficiently severe to suggest an abdominal crisis. Attacks of this sort have been observed on the administration of any type of viosterol or cod liver oil, even when taken in minute quantities. It would be interesting to know the mechanism of this type of sensitiveness.

Waldbott [5] reported severe asthma in a patient, which was caused twice by the application of a little cocaine in the nose for the purpose of local anesthesia. A scratch test with a 3 per cent solution produced erythema but no wheal. He also reported a case in which death almost immediately followed the application of procaine hydrochloride.

The toxicity usually attributed to local anesthestics, such as cocaine, is frequently, if not usually, a result of the hypersensitiveness of the patient to the drug, with the production of severe allergic shock. Illnesses which occasionally follow the injection of a local anesthetic

3. Abramowitz, E. W., and Noun, M. H.: Eczematous Dermatitis Due to Exposure to Chloral, J. Allergy **4**:338 (May) 1933.

4. Gordon, M. B., and Lieberman, H.: Idiosyncrasy to Viosterol, Am. J. M. Sc. **183**:784 (June) 1932.

5. Waldbott, G. L.: Asthma Due to a Local Anesthetic, J. A. M. A. **99**:1492 (Dec. 3) 1932.

with epinephrine are occasionally attributable to the epinephrine rather than to the local anesthetic. Some persons who are sensitive to the effect of epinephrine have marked tremor, a feeling of weakness and pounding of the heart after the use of the most minute quantities of this drug. These reactions can be stopped within from thirty to sixty seconds if the injection has been made in an extremity which permits the application of a rubber tourniquet above the site of application. This remedy would not apply in the case of nasal surgery. However, continued absorption of a drug such as cocaine could be reduced grossly if epinephrine were applied at the site of application of the anesthetic.

Harkavy, Hebald and Silbert [6] reported a study of thrombo-angiitis in persons who smoked tobacco excessively. They found that the intracutaneous injection of 0.1 cc. of a 1 : 10 solution of tobacco caused positive reactions within from ten to fifteen minutes in 83 per cent of the patients. In 13 of 20 patients passive transfer of sensitivity could be demonstrated. In many patients multiple sensitization was found. In the controlled cases, only 10 per cent of the subjects reacted to tobacco.

I might quote here an old saying that it is dangerous to change the habits of a man over 50. In the practice of medicine one is frequently confronted with the question of whether to advise the discontinuance of such habits as the excessive use of tobacco and coffee and the use of moderate or small quantities of alcoholic beverages which have been taken with regularity over a long period. Advice on this question should never be taken or given lightly. The same care should be used in relation to retirement from active business life or retirement from athletics. I have observed swelling of the nasal membranes which followed radical changes in habits such as those mentioned within from one to three months.

Ayres and Anderson [7] reported 4 cases of epidermophytosis in which there proved to be intolerance to Whitfield's ointment. It is interesting to mention this result in relation to the well known sensitiveness to acetylsalicylic acid, which occurs, according to Storm van Leeuwen, in about 10 per cent of persons with asthma.

Harrison [8] gave an interesting report on a patient who had urticaria during the menstrual periods. He made extracts from a pad used by the patient during the menstrual period and found that the extract caused

6. Harkavy, J.; Hebald, S., and Silbert, S.: Tobacco Sensitiveness in Thromboangiitis Obliterans, Proc. Soc. Exper. Biol. & Med. **30**:104, 1932.

7. Ayres, S., Jr., and Anderson, N. P.: Intolerance to Whitfield's Ointment as a Cause of Failure in the Treatment of Epidermophytosis, J. Allergy **4**:146 (Jan.) 1933.

8. Harrison, W. T.: A Case of Menstrual Allergy, J. A. M. A. **100**:738 (March 11) 1933.

positive skin tests and that the patient was completely relieved on treatment with the extract. This condition may be commonly found and is analogous to the sensitiveness of patients to their own breast milk.

Bullen [9] reviewed a large number of cases of chronic sinusitis and stated that only 12.25 per cent of the patients had asthma. He assumed that this indicated that chronic sinusitis per se is not a common cause of asthma. This, he stated, became evident on the finding that only 8.75 per cent of the patients had asthma and sinusitis at the same time.

PATHOLOGY

MacDonald [10] reported the observations at autopsy in 8 cases of bronchial asthma. The findings varied, of course, in different patients, but the usual observations, such as eosinophilia, hypertrophy of the bronchial musculature, hypertrophy and metaplasia, were noticed.

In my opinion, far too little attention has been given to the pathologic changes which occur as a result of asthma and so-called vasomotor rhinitis. These two symptoms in a growing child are serious and cause deformities of the face, nasal cells, chest and soft tissues which are destined to be permanent unless they are relieved before the period of growth is complete. Also, in evaluating the effect of a given method of therapy, the pathologic condition which has occurred as a result of allergy is not often taken sufficiently into account, and results are expected too quickly. In all cases of chronic allergy a certain length of time must be allowed for the restoration of the tissues, even after symptomatic relief has been obtained, and in many cases in which gross pathologic changes have entered the picture only partial relief can be expected by any method of therapy.

SYMPTOMS

Rinkel [11] gave a complete and very fair discussion of migraine. In the recent literature divergent opinions have been due, I think, to the indefiniteness of the term migraine and also to the lack of recognition of a typical familial migraine which is, without question, a clinical entity entirely apart from the factor of allergy, in the same sense that there is a familial epilepsy which is a clinical entity entirely apart from the factor of allergy.

No one denies that terrific attacks of headache resembling migraine and severe convulsions which are epileptic in appearance may occur as

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9. Bullen, S. S.: Incidence of Asthma in 400 Cases of Chronic Sinusitis, J. Allergy **4**:402 (July) 1933.

10. MacDonald, L. G.: The Local and Constitutional Pathology of Bronchial Asthma, Ann. Int. Med. **6**:253, 1932.

11. Rinkel, H. J.: Migraine, J. Allergy **4**:303 (May) 1933.

the direct result of a reaction caused by either allergy or physical allergy. This fact is accepted by all thorough students of medicine, and it is discussed completely and well by Rinkel. Rinkel's article should satisfy internists and neurologists who hesitate to accept the idea that familial migraine, periodic migraine and idiopathic family epilepsy are always or even in a majority of cases allergic in origin except when they occur in an allergic person and that either migraine or epilepsy can be precipitated in this case by a reaction of allergy.

Adamson and Sellers [12] reported an allergic study of 100 cases of epilepsy and stated that the occurrence of the manifestations of true atopy was higher in the antecedents than that found for normal persons, but that, on the other hand, it was lower than that reported in accepted atopic conditions, such as hay fever and asthma. They stated that the incidence of hay fever, asthma and eczema in the patients was low; also, that the occurrence of demonstrable atopic reagins in the serums of 11 per cent of the patients was somewhat higher than that expected in normal persons but was much lower than that reported in such accepted conditions of hypersensitiveness as hay fever and asthma. Their findings seem to harmonize better with common horse sense knowledge of medicine than those of investigators who try to prove that atopy is the real factor in the etiology of familial epilepsy. Their results seem to clearly indicate that the rather common condition, allergy, is an important factor in the precipitation of epileptic attacks in persons with true epilepsy.

Vaughan [13] may be added to the number of prominent investigators who discussed the relationship of allergy to pathologic conditions in the nose. He warned against deciding on surgical intervention until after a thorough allergic study has been made in suspected cases of allergy. He described the nasal picture of allergy, and the ease with which a differential diagnosis can be made between infection and allergy through the finding of gross quantities of eosinophils in nasal secretions. He also mentioned a finding which in the past has been deceptive—thickened membranes in the cells, which can be discovered through the use of iodized poppy seed oil 40 per cent and roentgen rays. He stated that an operation is not indicated in extrinsic asthma until after an attempt has been made to relieve the allergic condition. He found that two thirds of his patients with extrinsic allergy had been previously operated on and that surgical intervention had not relieved them. He stated that he had obtained the best results in allergic treatment in cases in which

12. Adamson, W. B., and Sellers, E. D.: Observations of the Incidence of the Hypersensitive State in One Hundred Cases of Epilepsy, J. Allergy **4**:315 (May) 1933.

13. Vaughan, W. T.: Some Rhinologic Aspects of Allergy, J. Allergy **4**:127 (Jan.) 1933.

there had been no local pathologic condition in the nose and that three fifths of the good results were obtained in cases in which there had been a definite pathologic condition of the nose for which no local treatment had been given. He asserted that a definite pathologic condition of the nose may indicate surgical treatment if more conservative measures fail to give relief.

The reasoning on which students of allergy, almost without exception, base this belief is that allergy is a general disease that involves not only the surface of the nasal membranes but also the membranes at the orifice of the nasal cells and those inside the nasal cells and causes swelling, hyperplasia and polypi. The swelling is caused by a deposit of antibodies from the blood in the membranes, which react on contact with an atopen (or an antigen). Surgical intervention cannot relieve this fundamental illness and is, therefore, a symptomatic measure. Piness was the first to emphasize forcibly that allergy is a nonsurgical disease of the nose, and he has been unanimously supported in this idea.

Rowe [14] called attention to the fact that allergy can cause disorders in the pelvic organs and that removal of the offending agents may be followed by relief. This is well worthy of repetition because of its importance. To this might be added the fact that pregnancy has a marked effect on allergy. Frequently a mother reacts to her own milk —as it is produced during pregnancy—with severe illnesses, such as urticaria, asthma, noninfectious coryza and other manifestations of reaction which may be so severe as to necessitate interruption of the pregnancy. Cases of this sort can frequently be relieved temporarily by the use of the breast pump.

Werley,[15] in discussing angina pectoris in relationship to allergy, stated that as vascular reactions, such as spasm of the arterioles, can occur as a result of allergy, there was no reason why coronary constriction could not be caused by the same condition. He cited investigators who recognized an association between indiscretion in eating and angina pectoris, which to him suggested that allergy to food is an underlying cause of the coronary condition. In a study of 62 cases of angina pectoris, history and skin tests indicated that 96.8 per cent of the patients were allergic to one or more foods. In a patient who was sensitive to seven common foods, correction of the diet relieved angina pectoris. In another patient, who was sensitive to thirteen foods, attacks of angina disappeared after adjustment of the diet. He mentioned other cases in which angina was associated with definite manifestations of allergy caused by sensitiveness to food.

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14. Rowe, A. H.: Uterine Allergy, Am. J. Obst. & Gynec. **24**:333, 1932.

15. Werley, G.: Is Allergy a Factor in Angina Pectoris and Cardiac Infarct? J. Allergy **4**:65 (Nov.) 1932.

Students of medicine accept with caution a new etiology for a clinical condition which has been studied so completely and widely as angina pectoris. I think that the time has come when both allergy and physical allergy will have to be recognized as additional and fundamental causes of angina pectoris. This type can be severe and dangerous. John Hunter stated that his life lay in the hands of any villain who tried to tease him.

PREPARATION OF EXTRACTS

Cooke and Stull [16] reported a new method of preparing and standardizing pollen extract, based on the theory that the active substance is a protein. Consequently the extract is standardized according to the protein nitrogen content. They also found that loss of the active substance was so great that standardization had to be renewed after a period of one year and that the restandardized extracts were as active as new extracts. Very old extracts were found to have lost both protein and the active substance and were apparently useless.

Unger and Moore [17] used an extracting fluid consisting of 5 per cent dextrose and 1 per cent cresol U. S. P. in distilled water. They stated that this fluid is as stable as glycerosaline extract and that it is much less painful when injected. They stated also that the clinical results seemed good so far as their studies had progressed.

Colmes [18] commented on the difficulty of standardizing therapeutic methods. He stated, and, I believe correctly, that the number of doses, the size of the dose or the total amount of pollen needed is not constant in any large number of cases. I think that the individualization of patients is the keynote to success.

Durham [19] repeated his usual yearly study of the geographic distribution of pollen for the preceding year. The highest count was obtained in the Middle West and exceeded 2,000 per cubic yard of air. The lowest count, obtained in Mexico City, was less than 100.

Bernton and Thom [20] again emphasized the importance of molds as an etiologic factor in allergy. They reviewed the history of 8 cases of definite nasal allergy. Three of the patients reacted only to molds.

16. Cooke, R. A., and Stull, A.: The Preparation and Standardization of Pollen Extracts for the Treatment of Hay Fever, J. Allergy **4**:87 (Jan.) 1933.

17. Unger, L., and Moore, M. B.: Studies on Pollen and Pollen Extracts, J. Allergy **4**:92 (Jan.) 1933.

18. Colmes, A.: Notes on the Treatment of Hay Fever, J. Allergy **4**:98 (Jan.) 1933.

19. Durham, O. C.: The Ragweed Season of 1932 in the United States, Canada and Mexico, J. Allergy **4**:105 (Jan.) 1933.

20. Bernton, H. S., and Thom, C.: The Importance of Molds as Allergic Excitants in Some Cases of Vasomotor Rhinitis, J. Allergy **4**:114 (Jan.) 1933.

Alternaria and Trichophyton were the only molds which these investigators used in testing which caused positive reactions. One patient had perennial symptoms due to contact with the organism Alternaria, which is commonly found in dust. Its distribution is so widespread and constant that patients who are sensitive to it have symptoms regardless of season or location. Sensitiveness to Trichophyton produces largely the same result because of the fact that it is a normal inhabitant of the skin.

DIAGNOSIS

Piness and Miller [21] compared the value of skin tests and elimination diets in the diagnosis of allergy in 4,589 cases. They concluded, with justice, that the elimination diet is a clumsy and cumbersome method as compared with skin tests. Furthermore, they stated, possibly with reason, that frequently elimination diets can hardly be carried out with anything short of a starvation diet. They thought, further, that many cases in which certain symptoms are relieved by elimination diets are not necessarily allergic unless they are associated with definite recognizable allergic syndromes, such as asthma, hay fever or urticaria. They concluded that, regardless of the heredity, the age of the patient at the onset or the age of the patient when tested, 80 per cent of allergic patients should give positive skin reactions and 70 per cent should react positively to foods. They stated that elimination diets in treatment, when based on the results of skin tests, are rational and reliable.

TREATMENT

Thomas and Touart [22] discussed thoroughly the significance of late skin reactions to vaccine. Thomas has for years devoted himself to this subject and has based his therapy on the results obtained by tests combined with clinical data. He has made wider observations of the phenomena which follow skin tests than other observers and has recorded these in a unique way and attempted to use them in outlining a therapeutic method which may be applied to the particular patient to be tested. He reported 71 per cent relief in a series of 300 cases of asthma associated with infection, obtained by treatment based on the interpretation of his tests. He also mentioned two types of general reaction which follow the use of the materials which he chose for treatment, one a relief reaction, the other a temporary exacerbation of the symptoms of which the patient complains. The

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21. Piness, G., and Miller, H.: Skin Tests in 4,589 Cases of Allergic Diseases, with a Criticism Concerning Elimination Diets, J. Allergy **4**:18 (Nov.) 1932.

22. Thomas, W. S., and Touart, M. D.: The Late Vaccine Skin Reaction, J. Allergy **4**:242 (May) 1933.

articles written by Thomas and his associates are almost too compact for satisfactory review, and for this reason I refer the reader to Thomas' original articles and also to some of the articles of Crowe on the vaccine treatment of arthritis. In treating arthritis, Crowe aimed to get a relief reaction instead of an exacerbation reaction and considered, in brief, that the optimum dose is that which gives relief for a period of from five to seven days. The dose is then gradually reduced, and a constant effort is made to produce relief reactions by the smallest possible dose. I have obtained this result in several cases of asthma in which I thought that infection was a factor and have obtained similar results in the so-called coseasonal treatment of hay fever and asthma due to pollen, as described by Vaughan, Phillips, Bernton and other observers.

Many investigators who have written articles on vaccine therapy or protein therapy have neglected the important item of fever. Fever relieves asthma. Even a rise from subnormal toward normal temperature may relieve a patient with asthma either temporarily or for a considerable period of time, and I believe that the temporary results obtained by many physicians are often due as much to the faint rise in temperature produced as to the specific effect of the agent used in therapy.

Fineman [23] reported on the treatment with renal cortex of 4 patients with chronic asthma. Three of the patients were sensitive to protein, and 1 was nonsensitive. Fineman noted a decided increase in strength in 3 of the 4 patients; their general condition was definitely improved. One patient showed marked improvement in asthmatic symptoms; 1, moderate improvement; 1 slight improvement, and 1, no improvement.

The use of cortin and other suprarenal substances should be tried, as they are discovered, in the treatment of bronchial asthma. I have tried cortin in a number of cases but not over so long a period as Fineman did. My results were not definite enough to justify a report.

Levine [24] reported benefit in the treatment of true vasomotor rhinitis by the subcutaneous injection of a 5 per cent aqueous solution of sodium iodide containing a trace of free iodine. Injections were administered at intervals of two or three days and gave satisfactory results. Negative results were obtained in the treatment of true rhinitis.

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23. Fineman, A. H.: The Use of Suprarenal Cortex Extract in the Treatment of Bronchial Asthma, J. Allergy **4**:182 (March) 1933.

24. Levine, M.: Free Iodine in the Treatment of Vasomotor Rhinitis and the Symptom Complex of Sneezing and Nasal Hydrorrhea; Preliminary Report, Laryngoscope **43**:39, 1933.

Maisel [25] reported a new method of immunization to poison ivy extract. One patient, who had been unsuccessfully treated by other means, experienced practical relief through the method finally used by Maisel, namely, exposing the skin to minute quantities of Rhus toxicodendron by a daily bath containing, at the beginning, 4 drops of an extract of Rhus toxicodendron to 10 gallons (37,854 cc.) of water. The concentration was raised at the end of twenty-five days to 30 cc. of the extract to 4 gallons (15,142 cc.) of water. During the period of administration, the patient occasionally had cutaneous reactions, but these were not severe. The method is considered harmless.

A great many conditions resembling Rhus toxicodendron dermatitis can be traced to sensitiveness to pollen oil, light, heat and other agents. This may account for some of the failures of Rhus toxicodendron therapy. Rhus toxicodendron therapy has been so successful in my hands, however, that failure to obtain a cure leads me to suspect an error in diagnosis or to assume that additional factors play a part.

Anderson,[26] after treating 60 patients with asthma with intratracheal injections of iodized oil, stated that improvement was gratifying in all cases and that in 24 cases the symptoms were relieved for a period of from one to five and a half years. He stated that the plugs of mucus in the smaller bronchi, which were shown by roentgen rays, were removed by injections of the oil, and that the patients who received intratracheal injections of the oil lost their hypersensitive response to known excitants.

The value of iodides in the treatment of asthma, especially of chronic perennial asthma, has been known for centuries. The conclusion that iodized oil or iodine removes hypersensitive responses to known excitants may perhaps be accounted for by Anderson's statement "that the number and scope of these cases must be extended before claims of a formal nature could be made in behalf of this method of treatment." It would be interesting if Anderson would report a series of cases showing the effect of iodized oil therapy on the positiveness of skin tests in cases of pollen allergy or a series of cases in which nasal and bronchial symptoms caused by pollen were treated with iodized oil. Perennial cases are inclined to vary both as to symptoms and skin tests, for no known reason.

Cunningham and Wolfe,[27] in a report on ten years' experience with therapy for pollen allergy, stated that it was difficult to compare the

25. Maisel, F.: Poison Ivy: A New Method of Immunization, J. Allergy **4**:35 (Nov.) 1932.

26. Anderson, W.: Some Observations on the Value of Intratracheal Injections of Iodized Oil for Bronchial Asthma, J. Allergy **4**:44 (Nov.) 1932.

27. Cunningham, T. D., and Wolfe, A. M.: Summary of Results of Ten Years' Hay Fever Treatment, J. Allergy **4**:48 (Nov.) 1932.

figures for one year with those for another, because of the yearly improvement in technical methods. Basing their conclusions on answers to questionnaires sent to 600 patients, 231 of whom sent replies, they reported 59.4 per cent relief for the first year of treatment, 66 per cent relief for the second year of treatment and 71.7 per cent relief for the third year. They stated that the patients who were completely relieved after one year of treatment were advised to discontinue treatment and that apparently they experienced two or more years of relative comfort, so far as hay fever was concerned, without treatment of any sort. Approximately one third of the patients with hay fever had asthma, and in these patients relief averaged 72.3 per cent.

Cunningham and Wolfe mentioned the gross difficulty in evaluating the results obtained by therapy for pollen allergy because of the gross variability of the pollen content of the air and the many different causative agents in different years. These sources of error have constantly prevented me from giving statistical accounts of the results of therapy. Such statistics are, of course, much more accurate if avoidance of pollen is practiced along with therapy and if patients are tested for sensitiveness to heat, cold and light and treated accordingly. Favorable results in the treatment of pollen asthma can be obtained in 90 per cent of the cases or more if the factor of heat and cold is taken into consideration and if too many pollens are not blown into a given district from distant sources.

Nelson, Rappaport and Welker [28] reported the results obtained by confining patients to a ward of eight beds which was supplied with filtered air. They stated that the commercial filters now manufactured are not 100 per cent efficient for the removal of pollen, but that filtration, which removes all but traces of pollen, relieves the symptoms of hay fever.

Symptoms recur on reexposure to pollen-laden air, regardless of the length of the patient's confinement to rooms with filtered air. The time required for reappearance of the symptoms depends on the concentration of pollen in the outdoor air.

Patients with pollen asthma were also relieved by this method of treatment, but a longer period of exposure to filtered air was necessary. In the more severe cases a still greater length of time was required. Nelson, Rappaport and Welker noticed that the temperature and moisture of the air were also factors.

Gay [29] studied the effect of air-conditioned atmosphere on hay fever and asthma caused by pollen during the pollen season. He found that the symptoms cleared up almost without exception within from

28. Nelson, T.; Rappaport, B. Z., and Welker, W. H.: The Effect of Air Filtration in Hay Fever and Pollen Asthma, J. A. M. A. **100**:1385 (May 6) 1933.

29. Gay, L. N.: The Treatment of Hay Fever and Pollen Asthma by Air-Conditioned Atmosphere, J. A. M. A. **100**:1382 (May 6) 1933.

four to twelve hours but that recurrences were likely when the patient was again exposed to the outside air. He recommended this method of treatment for patients who can have their homes or offices, or both, treated in such a way as to exclude the pollen from the air.

Prickman and Vinson [30] emphasized the usefulness of bronchoscopy in a limited number of cases of asthma and stated that the removal of mucus is immediately beneficial.

PHYSICAL ALLERGY

Weissenbach and Brisset [31] reported a case of urticaria caused by cold in which they could transfer sensitiveness to cold by the Prausnitz-Küstner method if the blood used for transfer was taken during a period of reaction to cold. In other words, if the skin of the patient was exposed to cold, a serum containing antibodies could be transferred to a normal person. These observers assumed that the antibodies are formed on exposure to cold and can be demonstrated in the serum by passive transfer but that ordinarily they are neutralized or absorbed.

I [32a] reported a study of this sort and referred to physical allergy as a new, very potent and often tragic cause of disease. These observations have been extensively confirmed by reports in the French and British literature, but in America the reactions in physical allergy are looked on chiefly as secondary phenomena caused primarily by some unfindable agent to which the patient is sensitive. The fact is that sensitiveness to heat and cold is a rather common illness and, peculiarly enough, is almost always overlooked by the patient as well as by the physician.

I [32b] reported cases of functional cardiac disorders, including extrasystoles, tachycardia (not paroxysmal) and angina pectoris, occurring in certain persons as a result of heat and effort or of cold. The symptoms could be precipitated at will by minute quantities of heat, effort or cold, as the case might be, and could be stopped almost immediately by the reverse factors. The patients were all benefited grossly by the controlled application of the agent responsible for the illness without resort to the remedies usually employed in the treatment of heart diseases.

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30. Prickman, L. E., and Vinson, P. P.: Bronchoscopy in Asthma, J. Allergy **4**:286 (May) 1933.

31. Weissenbach, R. J., and Brisset, J. P.: Urticaria from Cold, Ann. de méd. **32**:289 (Nov.) 1932.

32. Duke, W. W.: (*a*) Urticaria Caused Specifically by Action of Physical Agents, J. A. M. A. **83**:3 (July 5) 1924; (*b*) Relationship of Heat and Effort Sensitiveness and Cold Sensitiveness to Functional Cardiac Disorders Including Angina Pectoris, Tachycardia and Ventricular Extrasystoles, J. Allergy **4**:38 (Nov.) 1932.

Abstracts from Current Literature

Ear

THE FUNCTION OF THE SALIVARY GLANDS, ESPECIALLY AFTER RADICAL OPERATION ON THE MIDDLE EAR. F. ALTMANN and K. LÖWY, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:188 (March 21) 1933.

Altmann and Löwy studied the effect of destruction of the chorda tympani on the secretory function of the submaxillary and sublingual glands in persons who had undergone unilateral radical operation on the middle ear. This was done by determining the interval between the intravenous injection of iodine and its appearance in the secretion of these glands on the operative and on the nonoperative side and the duration of the secretion. It was first determined by the use of pilocarpine and atropine that the passage of iodine from the blood into the saliva is dependent on the parasympathetic nervous system. The investigations showed that neither a recent division of the chorda nor one dating several years back had a demonstrable influence on the secretion of iodine. This indicates a long duration of the functional capacity of the salivary glands concerned, which, on the basis of the investigations, cannot be attributed to the vicarious substitution of other nerves for the severed chorda. The results of the stimulation and the paralysis of the parasympathetic nerve endings have rather shown that the peripheral neuron beginning in the submaxillary or sublingual ganglion retains its normal function for years. These experiments indicate that the secretion of rhodan by the salivary glands (the subject of study by others, so far as it represents a functional activity of the glands, is governed by the same laws as the secretion of iodine. To attempt on the basis of the absence of such reactions to draw conclusions as to the condition of the chorda tympani or the character and the prognosis of an inflammatory process in the middle ear is futile, because even a certain division of the chorda tympani has an effect only under special experimental conditions.

EDITOR'S ABSTRACT.

MULTIPLE SCLEROSIS MISTAKENLY DIAGNOSED AS TUMOR OF THE RIGHT CEREBELLOPONTILE ANGLE: REPORT OF A CASE. W. BROCK and O. GAGEL, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:277 (April 4) 1933.

Brock and Gagel report a case in which the diagnosis of tumor of the cerebellopontile angle was made on the basis of severe headache, retrobulbar neuritis, severe bilateral baryecoia (especially on the right), vestibular hypo-excitability, facial paralysis on the right side and similar manifestations. No tumor was discovered at operation, and at necropsy, after death from bronchopneumonia, the diagnosis of multiple sclerosis was made. The nature of the lesions indicated a relatively early stage of the disease. Foci of disintegration of the myelin sheaths in the fibers of the emerging cochlear roots and in the region of the ventral cochlear nucleus explain the baryecoia. The portion of the acoustic nerve in the inner ear and the temporal bone was normal. In view of the similarity of the vestibular and cochlear symptoms of a tumor of the cerebellopontile angle and of multiple sclerosis the demonstration of the transitory nature of the disturbance of acoustic function may be the only guide to the diagnosis of multiple sclerosis. In the absence of this differential sign a mistaken diagnosis of tumor may be made and the patient needlessly subjected to a dangerous operation. Possibly in the case reported cochlear and vestibular examinations would have resulted in the correct diagnosis, although with the rapid evolution of the process it is not probable that the cochlear symptoms would have receded. The case shows how difficult the diagnosis of multiple sclerosis may be and the importance of repeated otologic examinations over a longer period of time in similar cases.

EDITOR'S ABSTRACT.

Is Labyrinthine Operation Indicated in All Cases of Labyrinthogenous Meningitis Following Acute Otitis Media? O. Voss, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:377 (April 4) 1933.

Voss states that while the existence of labyrinthogenous meningitis is generally considered an indication for operation on the labyrinth, a few cases have been reported in which meningitis of labyrinthine origin was healed by intervention on the middle ear alone; he has observed two such cases. In the first case, one of acute otitis media of eleven days' duration with diffuse purulent meningitis, he refrained from simultaneous intervention on the labyrinth because of a partial loss of cochlear and vestibular function. A thorough cleaning out of the original focus of infection in the mastoid and middle ear by epitympanic antrotomy resulted in an immediate remission of the symptoms and cure. In the second case the labyrinthogenous meningitis was relatively bland and in an early stage. Labyrinthine intervention was avoided in spite of a total loss of labyrinthine function, because of the severe disintegration of the mastoid and middle ear, including a deep epidural abscess. In both cases the cleansing out of the original infectious focus in the mastoid, completed by inclusion of the epitympanic recess in the surgical cavity, was performed thoroughly and painstakingly. The poor prognosis for labyrinthine operation in cases of acute otitis media and otitis interna with meningitis and the favorable results obtained by the author's method suggest that the indications for the labyrinthine intervention may need revision although the data are insufficient to permit a definite conclusion. Editor's Abstract.

Parietal Lobe Symptoms in Otogenous Contralateral Thrombophlebitis. G. Bodechtel and H. Richter, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:505 (March 20) 1933.

Bodechtel and Richter report the case of a man who suddenly experienced numbness and uncertainty of motion in the left arm four weeks after a severe catarrh which ended with baryecoia and pain in the left ear. Examination showed astereognosis, inability to find parts of the left half of the body with the right hand and hyperalgesia of the left side; total hemiparesis and analgesia of the left side developed later. Operation disclosed a thrombosis of the left sigmoid sinus extending to the torcular herophili, the place where the longitudinal sinus (in which the veins of the right hemisphere are joined) terminates. This indicated a progressive thrombosis. Contrary to expectations, necropsy showed no signs of thrombosis in the right parietal region. The only macroscopic changes in the right hemisphere were those of a diffuse purulent meningitis which affected both hemispheres. Microscopically, in addition to the meningitis, the cortex of the anterior central and parietal regions of the right hemisphere exhibited diffuse cellular disintegration in the three upper layers and strong proliferation of the glia cells. The authors assume that the thrombosis up to the torcular herophili led to a sudden vascular shock in the right hemisphere, accompanied by nutritional disturbance of the corresponding region. The injured tissue was much more receptive to the toxic influence of the meningitis than the rest of the brain which was not involved in the primary functional circulatory disturbances. Editor's Abstract.

The Pathogenesis of Cholesteatoma. W. Albrecht, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:517 (March 20) 1933.

Albrecht considers the persistence of embryonic tissue in the epitympanic recess the prerequisite for the tendency to proliferation of the squamous epithelium which is the main cause for the development of a cholesteatoma. This contradicts the opinion expressed by Lange in his article, entitled "Deeply Retracted Pars Flaccida and Cholesteatoma" (*Ztschr. f. Hals-, Nasen- u. Ohrenh.* **30**:575 [July] 1932; abstr., Arch. Otolaryng. **17**:110 [Jan.] 1933). Lange considers the connective tissue of the attic, found in cholesteatoma, newly formed scar tissue of inflammatory origin. He thinks that atypical connective tissue can never be demonstrated in

tympana totally free from inflammation. The author considers this explanation of little value in view of the fact that otitis occurs in about 60 per cent of nurslings and frequently in patients at other ages. He thinks that the persistence of the original lumen in the proliferated connective tissue points to an inflammatory origin, and that its absence permits the certain recognition of embryonic tissue. The connective tissue of inflammatory origin has a much firmer structure than the loose structure of the embryonic tissue, and the squamous epithelium cannot penetrate it. Embryonic tissue persisting in the attic is a loose, large-meshed tissue with sparse fibers which offers little resistance to the proliferations of the irritated squamous epithelium in the region of the pars flaccida, which is consequently able to spread. The fact that in 80 per cent of cases of cholesteatoma there is a compact mastoid process supports this view. The compact mastoid corresponds to a mucosa predisposed to cholesteatoma. This predisposition is based on the persistence of embryonic tissue. The development of a cholesteatoma and its size depend on the location and extent of the embryonic tissue.

EDITOR'S ABSTRACT.

CELLULAR REACTIONS IN THE LABYRINTH TO CHANGES OF AIR PRESSURE. C. F. WERNER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:544 (March 20) 1933.

Werner studied the effect of changes in air pressure on the labyrinths of guinea-pigs by fixing the labyrinthine specimens histologically at different pressures immediately after decapitation of the animals. He states that increases or decreases of air pressure effect regular changes in the height of the epithelium and in the cellular picture of the macula sacculi. These changes are not explainable mechanically but depend on reactions of the living cells. They are lacking in the macula utriculi. The changes in the macula sacculi, therefore, evidently depend on a specific sensitivity and are perhaps an expression of a functional relation of this macula to the atmospheric pressure. This would accord with the fact that as yet no other function has been demonstrated for the macula sacculi in mammals.

EDITOR'S ABSTRACT.

THE PATHOGENESIS OF OTOGENOUS PARAPHARYNGEAL ABSCESSES. D. M. RUTENBURG, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:25 (April 12) 1933.

One of the ways in which infection may be transmitted from the ear to the parapharyngeal space is via the lymph channels. Another way is by direct spread through the digastric fossa and along the digastric muscle to the retromaxillary fossa where the parotid gland is located. From there the infection may spread through the parapharyngeal space to the exterior of the tonsil. The other direct route is via the mastoid cells around the labyrinth or the floor of the tympanic cavity, which sometimes extend to the inferior petrosal sinus under the cochlea or along the carotid canal to the apex of the pyramid. Pneumatic cells in the anterior wall of the tympanic cavity may also develop toward the pyramid. The pus filling these cells may break through to the outside and pass along the eustachian tube to the parapharyngeal and retropharyngeal spaces. The spread of a carious process to the first cervical vertebra and from there to the parapharyngeal space is a fourth, rare form of pathogenesis. Rutenburg reports a case in which the infection followed the route of the mastoid cells and the eustachian tube, causing an abscess in the parapharyngeal space. From there the suppurative process spread beyond the parotid gland and the incisura Santorini and broke out in the posterior inferior portion of the external auditory meatus. This same inflammatory process caused extradural trigeminal neuralgia and the syndrome of Gradenigo. The author calls attention to the fact that following the injection of a 10 per cent suspension of bismuth in oil into the fistula as a contrast medium, the suppuration stopped, the temperature became normal and recovery was rapid. An analogous instance of the favorable effect of injection of bismuth into a fistula was recently observed by him.

EDITOR'S ABSTRACT.

ROENTGENOGRAPHY OF THE TEMPORAL BONE AND ITS CLINICAL SIGNIFICANCE IN AURAL DISEASE IN EARLY CHILDHOOD. S. I. WULFSON, A. L. KAPLAN and E. I. IAROSLAWSKY, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:88 (April 12) 1933.

Wulfson and his associates think that Sonnenkalb's method is best for roentgenography of the temporal bone in infants and small children. The head of the child is placed on the casette and loosely strapped, and the trunk is held in place by an assistant. An exposure of from one third to three fourths of a second is sufficient. The author has examined by this method 110 temporal bones (55 patients), including normal bones and bones with acute, subacute or chronic processes. In the examination of children up to 4 years of age, in normal cases the pneumatization periods of Wittmaack could be followed. A pathologic condition in any portion of the temporal bone becomes manifest in the roentgenogram. While on the healthy side the honeycombed structure of the antrum and periantral region is easily recognized, on the diseased side these areas are poorly differentiated or obscured. Zones of lesser density appear when the bone is attacked. When both sides are diseased and the roentgenographic images are similar, diagnosis may be less easy and careful comparison with the clinical data is important. The authors conclude that roentgenography of the temporal bone is valuable for the study of the development of the bone and for the recognition of pathologic processes exhibiting no definite clinical symptoms, and that serial examination in acute cases gives valuable diagnostic and prognostic aid.

EDITOR'S ABSTRACT.

PSYCHIC INFLUENCE ON PATIENTS WITH ORGANIC BARYECOIA. W. ROGGENKAMP, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:94 (April 12) 1933.

Roggenkamp states that while suggestion therapy in patients with organic baryecoia did not bring about any improvement of hearing which could be demonstrated by tests made with the oto-audion or by other methods, it did influence their attitude toward their impaired faculty. Eighty per cent of the patients reported an improvement in hearing which manifested itself in such ways as hearing traffic signals which formerly were unperceived, hearing the clock strike and being better able to understand their friends in conversation. The author's purpose is not to recommend this therapy but to draw a line between appearance and reality.

EDITOR'S ABSTRACT.

ROENTGENOLOGIC DIAGNOSIS AND THERAPY OF CONGENITAL OSSEOUS ATRESIA OF THE EXTERNAL AUDITORY MEATUS. R. MITTERMAIER, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:213 (July) 1933.

Mittermaier successfully constructed an artificial auditory canal in the left mastoid antrum of a girl, aged 20, with bilateral congenital imperforation of the auditory meatus. The left side was chosen for operation because the head of the malleus was visible on the roentgenogram of this side while it was missing on the right side. The degree of malformation, according to statistics, is usually less in cases in which the malleus and incus are present. In most cases in which operation has been successful ossicles were present; improvement was slight or lacking when they were absent. The patient's hearing on the side operated on was greatly improved for vowels of the lower register (*C* to *C 2*), and she was able to understand conversation at a distance of 5 meters, as contrasted to from 1 to 2 meters prior to operation. Operation is indicated only if two conditions are fulfilled. In the first place, the baryecoia must be primarily a defect of conductivity. This can be determined by functional tests, especially the audiometer curve and the test for vowel hearing. If there is a pronounced labyrinthine baryecoia, the creation of an air passage is ineffective. In the second place, the

presence not only of an antrum but of the tympanic cavity with its organs must be considered, and the ossicles, at least the head of the malleus, should be roentgenographically demonstrable. The author recommends the axial exposure of the temporal bone for this purpose.

EDITOR'S ABSTRACT.

Pharynx

THROMBOPHLEBITIC SEPTICEMIA. W. ALBRECHT, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **134**:258 (April 4) 1933.

Albrecht reports two cases of posttonsillitic septicemia. In one case a septic thrombosis of the jugular vein developed which extended centrally to the superior thyroid vein and peripherally to the torcular herophili. The patient's life was saved after extensive opening of the vein, although metastases had developed in the pleura. The patient in the other case died of severe sepsis and numerous metastases in the lung, although several hours after the first chill the facial vein was ligated, the jugular vein was excluded by excision of a large piece and the parapharyngeal space and peritonsillar tissue were opened. No thrombi were found in the veins; histologically there were signs of an active endophlebitis. Thrombus formation is considered a favorable reaction of the venous wall, the absence of which resulted in death in one of the cases. This may have been due to the greater virulence of the organism, but it may also have been due to a weaker individual capacity of the venous wall for protective reaction. The author thinks that in asthenic persons the sinus walls are frequently thin and have little resistance to infection. This may also be true of the venous walls and may be of equal importance in the development of a sinus phlebitis.

EDITOR'S ABSTRACT.

MYIASIS IN CONNECTION WITH CHRONIC TONSILLITIS. L. JENEY and F. LÖRINCZ, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:157 (June 15) 1933.

Jeney and Lörincz report that the larva of a fly belonging to the family Calliphorinae was found in the tonsil of a girl during tonsillectomy for chronic tonsillitis. No intestinal myiasis could be demonstrated to explain the finding of the larva. Histologically, no evidence of destruction of tissue by the larva could be found. It appears that the larva of a fly that causes semispecific myiasis accidentally penetrated the chronically inflamed tonsil, where it found nourishment without causing special damage to the host. No case of tonsillar myiasis could be found in the literature.

EDITOR'S ABSTRACT.

PERITONSILLAR ABSCESS AND TONSILLECTOMY FOR ABSCESS (WINKLER'S OPERATION). A. LINCK, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:227 (July 8) 1933.

Linck again affirms that the treatment of a peritonsillar abscess should consist in immediate tonsillectomy instead of incision of the abscess. Only by removal of the primary seat of infection, the tonsil, can the recurrence of peritonsillar abscesses be prevented. The substitution of tonsillectomy for incision of the abscess not only avoids a second prophylactic intervention but constitutes the best surgical therapy of the abscess. Resection of the tonsil, which usually forms the front wall of the abscess, effectively drains the abscess whether it is a small concealed one or a large ripe one. No spreading with the dressing forceps and no tamponade are necessary. Only in extremely rare cases in which the abscess has developed in the muscular layer are trial puncture and incision necessary. The exposure of the posterior wall of the abscess by tonsillectomy insures rapid healing and guards against the spread of the suppuration to the cervical veins. The danger of propagating the infection by tonsillectomy during acute inflammation of the tonsillar region has been disproved by experience. The opening of lymph channels and tissue spaces occurs just as much in incision of the abscess. More than 200 tonsillectomies for abscess in the author's clinic have not resulted in a single complication due to the intervention. The danger of spreading the infection,

attributed to local anesthesia, is avoided by a combination of regional superficial anesthesia from the exterior and superficial infiltration anesthesia from the interior. Hemorrhages are less frequent in tonsillectomy than in incision and are more easily stopped by acupression than by the tamponade used in hemorrhage from incisions.

EDITOR'S ABSTRACT.

CAVERNOUS ANGIOMA OF THE TONSIL. I. FRAENKEL, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:268 (July 8) 1933.

Fraenkel reports a case of cavernous angioma of the left tonsil in a man, aged 29. Only seven other cases have been reported. There was an anamnesis of frequent tonsillitis and occasional hemorrhages from the throat after violent coughing. The left tonsil was greatly enlarged. It was dark red and nodular, resembling a blood-soaked sponge. A diagnosis of tonsillar angioma was made, and the tonsil was extirpated, together with the adherent glossopalatine arch, after ligation of the plainly visible vessels. The tumor was 5 by 2.5 cm., dark brown and rough. The cut surface exhibited a honeycomb structure. A nodule of tonsillar tissue 0.5 cm. in diameter was found in the center, as well as a few nodules the size of a pinhead; microscopically, these nodules appeared encapsulated in a thin layer of connective tissue. In place of the reticulo-adenoid tissue usually found in the tunica propria, there was cavernoma tissue consisting of a coarse reticulum made of trabeculae surrounding alveolae filled with blood. The author describes the histologic picture in detail.

EDITOR'S ABSTRACT.

SPONDYLARTHRITIS OF THE CERVICAL VERTEBRAE AS A CAUSE OF DYSPHAGIA. C. WIETHE, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:54 (March) 1933.

Wiethe reports three cases of dysphagia caused by spondylarthritis of the cervical vertebrae, in each of which the mechanism of the dysphagia was different. In the first case, the act of swallowing caused pain radiating into the shoulder through irritation of the cervical nerves. In the second case, there was dysphagia with paresthesia produced by a large exostosis. In the third case, there was unilateral paralysis of the soft palate with unilateral hypesthesia of the pharynx through pressure on the pharyngeal plexus. In a case in which minor examination discloses no cause for dysphagia, roentgenography may discover the cause of the disturbance, as these cases demonstrate.

EDITOR'S ABSTRACT.

A CASE OF AGRANULOCYTOSIS CURED BY TONSILLECTOMY. W. KINDLER, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:85 (May) 1933.

Kindler differentiates between the pure form of agranulocytosis, which is of unknown cause, and the symptomatic form, which has a known toxic or infectious basis and leads to a syndrome of agranulocytosis. He reports a personally observed case of symptomatic agranulocytosis associated with a septicemia of tonsillar origin. The etiologic diagnosis of the case was complicated by the fact that the patient had just received a course of antisyphilitic treatment and that either the syphilis or the neoarsphenamine might have caused the agranulocytic syndrome. Tonsillectomy and drainage of a retrotonsillar abscess resulted in an immediate disappearance of the necroses and improvement of the clinical symptoms; the agranulocytic changes in the blood persisted for several months before gradually receding. The author thinks that the neoarsphenamine therapy, through injury to the leukopoietic system, paved the way for the agranulocytosis. The occurrence of a severe pneumococcic tonsillitis with abscess formation caused the clinical manifestations of the agranulocytosis in the form of necroses of the mucosa associated with a picture of severe sepsis.

EDITOR'S ABSTRACT.

Larynx

PRECANCEROUS DISEASES OF THE LARYNX. S. ZOLTÁN, Monatschr. f. Ohrenh. **67**:597 (May) 1933.

Zoltán discusses precancerous diseases of the larynx on the basis of observations of numerous patients, often over a long period of years. Pachydermic processes of the larynx frequently form the basis for the development of cancer—the soft forms as well as the hyperkeratotic forms. Among laryngeal papillomas the hemorrhagic and hyperkeratotic forms are to be regarded as precancerous changes. The productive form of tuberculosis, although associated with epithelial proliferation, cannot be considered the starting point of a carcinoma. Lupus and gummas cannot be regarded as primary changes in precarcinosis. The cicatricial processes associated with them, however, prepare the ground for cancer. The cause of the development of a carcinoma following trauma is to be found in the processes of epithelial regeneration.

EDITOR'S ABSTRACT.

CHRONIC TUBERCULOSIS OF THE LARYNX IN PATIENTS WITH ABACILLAR SPUTUM. F. I. DOBROMYLSKI, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:140 (June 8) 1933.

Dobromylski found that 9 per cent of patients with tuberculosis of the larynx had abacillar sputums; of these, 4.6 per cent had a chronic infection and 4.4 per cent an acute infection. The laryngeal infection in these cases is probably hematogenic. Abacillar sputum in the chronic forms of tuberculosis of the larynx is found under two conditions. In one third of the cases the pulmonary process is inactive or calcification has occurred; in two thirds of the cases there is a disseminated chronic or, rarely, a subacute process with a tendency to fibrosis. The diagnosis of the cases with the disseminated form is readily made, but in those in which there is no active pulmonary process the diagnosis is difficult, especially if there are only calcified foci present. In these cases it is also difficult to prove that there is a genetic relation between the laryngeal and the pulmonary process. If possible, a biopsy should be performed. In addition to roentgenography, the tuberculin test is valuable as a diagnostic measure. In almost all cases allergy was slight. In both groups, especially the second group, surgical therapy should be attempted only after a preliminary period of therapy for strengthening the general resistance. The subacute disseminated forms may be treated surgically only with the greatest caution. The prognosis for the patients of the first group is favorable, not only as to life, but as to ability to work. The patients of the second group, except for those with the subacute disseminated forms, may remain capable of work for quite a long time. The latter, because of the return of symptoms once or twice a year, need to interrupt their work with prophylactic rest periods.

EDITOR'S ABSTRACT.

PARALYSIS OF THE RECURRENT NERVE. L. RÖTTGER, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:98 (May) 1933.

On the basis of personal experiences as well as other investigations, Röttger refutes the commonly accepted theory that in paralysis of the posterior crico-arytenoid muscle the vocal cord remains fixed in the median position. The clinical picture of paralysis of the posterior crico-arytenoid muscle is instead characterized by movement of the vocal cords; there is an inward movement up to the median line and an outward movement up to the intermediate position. These limited movements are due to the alternate contraction and relaxation of the adductor muscles. The action of the posterior crico-arytenoid muscles consists only in the opening of the glottis from the intermediate position outward; it effects an abduction which exceeds that of quiet breathing. The author further contends that the diagnosis of paralysis of the posterior crico-arytenoid muscle on the basis of fixation in the median or paramedian position is not tenable; the condition must be considered as a paralysis of the recurrent laryngeal nerve. Clinically, an injured adductor function can be well differentiated from the isolated loss of posterior crico-arytenoid function and from total loss of function of the muscular apparatus.

EDITOR'S ABSTRACT.

Nose

HEADACHE AND PAIN IN INFLAMMATION OF NASAL SINUSES. WILFRED HARRIS, Brit. M. J. **1**:551 (April 1) 1933.

Harris comments on the difficulty of distinguishing the cause of headaches which are amenable to physical intervention from those which are not. The periodicity of frontal sinusitis, commencing at 9 or 10 a. m. and lasting to late afternoon, may simulate a migraine or may even be mistaken for paroxysmal neuralgic tic. Two cases of abscess of the frontal lobe following infection of the frontal sinus are reported.

The radiation of pain far beyond the territory of the actual nerve trunk affected makes diagnosis difficult. For example, an aching molar tooth may cause referred pain upward or downward to the upper or lower jaw, or to the ear, and, if unrelieved, the pain may spread to the neck and down the arm to the fingers of the same side. Harris emphasizes that in high-strung neurotic persons referred pain spreads more widely and readily, but it is important to remember that referred pain does not spread across the middle line. Therefore, when general headache is complained of in inflammation of a sinus it may be assumed either that general toxemia is present or that there is bilateral involvement of the sinuses. The theory that sensory painful impressions may be carried centrally along sympathetic fibers is not acceptable to the author.

Facial psychalgias are fairly common and are frequently mistaken for and treated as tic douloureux. True trigeminal neuralgia may have its origin from an infected antrum. Two common forms of pain in the face which may be mistaken for that of chronic sinusitis are: (1) migrainous neuralgia and (2) chronic neurosis of the jaw. In the former, there is a regular periodicity, every few weeks, perhaps accompanied by nausea and lasting for from twenty-four to forty-eight hours. In addition there may be a history of migraine in near relatives and frequently typical hemicranial attacks with vomiting and teichopsia.

Chronic neurosis of the jaw is an atypical neuralgia, almost limited to women of reproductive age and mainly psychalgic in origin. The pain is constant, affecting mainly the cheek and maxillary region on one side only, and is unaffected by movements of eating or by handling the face. It is always present during the waking hours, and as a rule sleep is not interfered with. Sometimes flushing and even slight swelling of the cheek may be complained of. Local treatment for this condition is to be discouraged. Many of the cases can be traced to some nervous shock. In regard to the latter group, the author remarks: "The frequency of cranial, and especially facial psychalgias, may be explained by the natural reference of one's 'ego' or individual personality to the region of the eyes and face, which more than any other part of the body express the emotions." The observation is made that among the blind St. Dunstan's men, who are especially liable to neurasthenia, their neurasthenic headaches are always referred to the cranium, and no case of facial psychalgia has been observed among them.

FERGUSON, Niagara Falls, N. Y. [ARCH. NEUROL. & PSYCHIAT.]

SEPTICEMIA AFTER ACUTE SUPPURATION OF THE ACCESSORY SINUSES. W. UFFENORDE, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:1 (April 12) 1933.

Uffenorde reports the case of a man with a typical picture of septicemia for which no explanation could be found by careful internal examination. A roentgenogram showed a zone of increased density in the right maxillary sinus with great swelling of the mucosa. On puncture a mucopurulent secretion was obtained. In radical operation on the maxillary, ethmoid and sphenoid sinuses on the right side, after removal of the diseased mucosa from the maxillary sinus, another cavity was found behind it, separated from the first one to a great extent by a bony septum. This cavity and the ethmoid sinus were cleaned out; the sphenoid sinus was found free, and the frontal sinus was opened and probed. The second day after operation the temperature dropped rapidly and complete recovery soon followed. The rapid

disappearance of the septicemic symptoms in response to the radical operation on the sinuses, in the absence of any other cause for the septicemia, definitely indicates that the septicemia originated in the sinus infection. The author considers the case one of sepsis without thrombosis. He thinks that radical intervention with exposure of the entire infectious focus is the only adequate procedure in cases of this kind. In this case the rare second chamber of the maxillary sinus would have been overlooked if this had not been done.

EDITOR'S ABSTRACT.

METAL PLATE SUTURE OPERATION FOR OZENA: RESULTS OVER A TEN YEAR PERIOD. V. HINSBERG, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:293 (July 8) 1933.

Hinsberg reports the results obtained with his operation for ozena in 152 cases in which periodic postoperative examinations were made. In the cases observed for from one to eight years, the author witnessed 68 per cent cures, 21 per cent improvements and 11 per cent failures. Patients were considered cured if the fetor and crusts disappeared completely, the mucosa became succulent and the nasal lumen was no longer significantly enlarged. They were considered improved if the fetor and crusts disappeared completely from one side of the nose while persisting so slightly on the other side that they could be controlled by irrigation and the patient felt practically free from symptoms. When the fetor and crusts were improved only slightly or not at all the operation was considered a failure. The operation aims to constrict the nasal lumen by bringing the conchae in close contact with the septum. It consists essentially in mobilizing the medial walls of the maxillary sinus by chiseling through them, transposing them medially and fixing them in place against the septum by the application of metal plates. The metal plates are held tightly against the maxillary surface of the lateral nasal walls by a wire previously passed through the nasal wall and inferior concha of one side, through the septum and through the concha and nasal wall of the other side. The plates are made of magnesium, which is resorbed within from six to eight weeks, during which period the artificial fractures are healed in the correct position. Postoperative treatment consists only in cold packs on the cheeks, which are usually swollen, daily irrigation of the nose and careful opening of the oral wound after two or three days to see if a suppuration of the maxillary sinus has developed and, if so, to treat it. Removal of the wire through the nose ten weeks after operation is the last step. The most frequent complication was suppuration of the maxillary sinus; it usually yielded quickly to treatment. A few cases of otitis media, dacryocystitis and abscess of the septum were observed. In a few cases the metal plate cut through the nasal wall, either because the wall had been injured or because the nose was too wide and the resistance of the walls too great. The operation is indicated if conservative methods such as irrigation and vaccination have failed and the symptoms are severe enough to be a serious disturbance to the patient socially or professionally. The patient should be warned of its inconveniences. It is contraindicated in patients much over 30 years of age and in those with general disease or disease of the frontal sinus. High grade atrophy of the mucosa or an extremely wide nasal lumen is an impediment to cure.

EDITOR'S ABSTRACT.

Miscellaneous

ROENTGENOLOGIC INVESTIGATIONS OF THE HEALING OF SKULL FRACTURES. ERNA LINDEMANN, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:25 (May 17) 1933.

Lindemann states that roentgen examination of thirty skulls with fractures of variable localization corroborates the reports in the literature that longitudinal fractures and trepanation defects of the skull practically always heal with the formation of connective tissue. Defects, which do not exceed the size of a bean, show a tendency to bone regeneration. Small, fine fissures sometimes heal with bone formation, so far as this can be demonstrated roentgenologically. Roentgen exam-

ination is of great value for the clinical evaluation of the fractures of the skull. Only by roentgen or anatomicopathologic examination is it possible to determine whether a fracture of the skull has healed by fibrous or by osseous growth.

EDITOR'S ABSTRACT.

ACCIDENTS AFTER ELECTROCOAGULATION OF MALIGNANT TUMORS. O. KAHLER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:87 (May 17) 1933.

Kahler has used electrocoagulation combined with irradiation (before or after or both) with favorable results in the treatment of tumors of the oral cavity, the mesopharynx and particularly the supramaxilla, but he reports a few accidents as a warning of the dangers encountered with this method. In the first case the patient died five hours after electrocoagulation of an adenocarcinoma of the maxillary sinus and the sphenoid sinus. Large fat emboli were found in the lungs and the kidney at necropsy. The tumor had extended into the fat of the cheek, and it had been necessary to coagulate it. Hemorrhage from a few vessels occurred, which stopped after coagulation. The author thinks he used too strong a current in proportion to the size of the electrode; according to von Seemen, this leads to rapid surface coagulation and rupture of the blood vessels below. In regions rich in fat coagulation must be undertaken cautiously with a current of small intensity to prevent hemorrhage and the danger of fat embolism. Besides the pulmonary embolus, the shock resulting from the circulatory disturbances produced by the embolism may have been a contributory cause of death. In the second case, seven weeks after electrocoagulation of a carcinoma of the supramaxilla which had invaded the orbit, a meningitis starting from a sequestrum of the orbital roof rapidly led to death. Other cases of meningitis after electrocoagulation of the bone adjacent to the dura have been reported. Electrosurgical treatment of diseased bone near the dura appears to be contraindicated. If an injury of the bone during electrocoagulation seems unavoidable, the bone should be removed with a chisel. In the third case, eight days after coagulation of a carcinoma of the left side of the tongue, there was a severe hemorrhage from the tongue which could not be completely arrested by ligation of the common carotid. Death resulted from encephalomalacia. Necropsy revealed that the hemorrhage had not occurred on the left side but on the right side following the development of a collateral circulation. The ligation of the two lingual arteries preceding the operation was performed in the typical place between the fibers of the hypoglossus. While the left lingual artery was ligated anterior to the branching of the sublingual artery, the right lingual artery was ligated posterior to the point where the sublingual artery branches. A collateral circulation could also develop without an abnormal anastomosis of the sublingual artery, as the latter sometimes arises from the submental artery.

EDITOR'S ABSTRACT.

BACTERICIDAL ACTION OF SPINAL FLUID. HESSE, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:105 (June 8) 1933.

Hesse studied the bactericidal action of human spinal fluid under normal conditions and in cases of meningitis and also the bactericidal action of normal and of pathologic spinal fluid of rabbits. The plate-counting method used in testing for the bactericidal properties of blood was employed. Autogenous bacteria were used when possible, chiefly hemolytic streptococci and Streptococcus mucosus, rarely pneumococci, staphylococci and other bacteria. Normal human spinal fluid sometimes possesses bactericidal properties; immediately after the onset of meningitis the spinal fluid almost always possesses bactericidal properties. The spinal fluid of rabbits is always bactericidal under normal conditions and almost always after the onset of meningitis. The bactericidal properties of the blood and the spinal fluid are not always parallel. It appears that in meningitis, antibodies are formed in the spinal fluid; they do not have to originate in the blood. The addition of complement to the spinal fluid is not necessary to determine the presence of bactericides; it may give rise to wrong conclusions. From the standpoint of

prognosis, the results indicate that the absence of bactericides in the spinal fluid immediately after the onset of meningitis is unfavorable, as they were almost invariably present at this period in the cases which cleared up. The existence of bactericidal properties at this period is not a guaranty of a favorable outcome of the meningitis, as they may rapidly disappear. In one or two cases bactericidal properties were found to be present almost immediately before the fatal termination of the disease.

EDITOR'S ABSTRACT.

METHODS OF STEREOROENTGENOGRAPHY. W. BERGER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:282 (July 8) 1933.

Berger considers stereoroentgenography a valuable diagnostic aid in otorhinolaryngology. With the use of Osterwald's apparatus, stereoroentgenograms at the exposure angles of Schüller and Stenvers can be made more easily than single exposures. An increased amount of film is not needed. Berger also uses Osterwald's apparatus for taking stereoroentgenograms of the frontal and ethmoid sinuses. This is best accomplished with the rays passing in the occipitonasal direction. Screening off the opening of the tube has the same effect as using the Bucky diaphragm. The distortion of perspective and of spatial relationship which may occur in stereoroentgenograms does not detract from their diagnostic value. Clinically, it is important only that the stereoscopy be qualitatively correct; it is sufficient that the spatial impression permit the anatomic relations between the parts to be demonstrated. It is not necessary to overcome this distortion by Hasselwander's two mirror stereoscope as suggested by certain critics of stereoroentgenography.

EDITOR'S ABSTRACT.

TREATMENT OF DILATATION OF THE ESOPHAGUS. A. GREIFENSTEIN, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:143 (May) 1933.

In two cases of severe idiopathic dilatation of the esophagus, Greifenstein obtained favorable therapeutic results with the use of a permanent catheter. It consists of an esophageal catheter, 20 cc. in length and 14 mm. in diameter, vulcanized at one end to a nasal catheter from 4 to 6 mm. in diameter; it is closed at its distal end. The esophageal catheter has numerous large perforations arranged spirally. It is introduced under roentgenologic control so that half of it lies in the stomach and the other half in the esophagus; the thin oral end of the catheter emerges through the nose. The introduction of the catheter was followed by a pronounced improvement of all symptoms due to stasis and esophagitis; sensations of pressure and burning ceased, and there was no more choking or vomiting. In one case the catheter was employed for one month; in the other case, for three months. A significant gain in weight was observed in both cases. The roentgenologic picture did not correspond to the clinical improvement noted. Examination one year after the conclusion of treatment found the patients free from symptoms; increase in weight had been maintained or increased. In two other cases of moderate esophageal dilatation with pronounced cardiospastic manifestations, paravertebral anesthesia was employed as a therapeutic measure; the results warrant further application of this method.

EDITOR'S ABSTRACT.

CASE OF ACTINOMYCOSIS OF THE TEMPORAL BONE EXAMINED HISTOLOGICALLY. G. THEISSING, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:165 (May) 1933.

Theissing reports a case of actinomycosis of the temporal bone which was not diagnosed until autopsy. Histologic examination indicated that it spread from a pharyngeal port of entry, along the large vessels and nerves to the base of the skull and the right petrosa, leading to a severe actinomycotic meningitis with circumscribed suppurative foci between the cerebellar convolutions. From a thrombosis of the bulb of the jugular vein a metastatic infection of the lung developed. Besides severe osteomyelitis of the pyramid there was an otitis media originating via the eustachian tube. The clinical course was slow, with relatively few symptoms.

EDITOR'S ABSTRACT.

Society Transactions

AMERICAN OTOLOGICAL SOCIETY

ABSTRACT OF TRANSACTIONS BY ROBERT SONNENSCHEIN, M.D., CHICAGO

Sixty-Sixth Annual Meeting, May 8-9, 1933, Washington, D. C.

GEORGE L. TOBEY, JR., M.D., *President, in the Chair*

Morning Session, May 8, 1933

PRESIDENTIAL ADDRESS. DR. GEORGE L. TOBEY, JR., Boston.

Dr. Tobey called attention to the fundamental pioneer work in otology done by St. John Roosa, Clarence Blake, Buck, J. Orne Greene, Alexander Randall, Gruening, Whiting, McKernan, Jack, Dench and many others. Among the other achievements of the American Otological Society have been the foundation of the Research Committee on Progressive Deafness, the compilation and publication of a complete bibliography on otosclerosis and a study and presentation of the ossification centers of the internal ear, with the demonstration of the blood supply of the labyrinth. This society has seen the invention of the audiometer, the standardization of tuning forks, the true evaluation of the caloric and rotation tests and, to a large extent, the researches made by McNally and others, together with the clinical work of Lewis Fisher. "The treatment of traumatic palsy promises to have been solved by the monumental work of Dr. Charles Ballance and Dr. Arthur Duel."

Dr. Tobey calls attention also to the studies on the cellular elements of the petrous pyramid and to the study of the dynamics of the cerebrospinal fluid which has demonstrated the value of the manometer test as a diagnostic procedure in lateral sinus thrombosis. The genetics of otosclerosis have been studied, and Wever and Bray in their experiments have demonstrated a microphonic effect detected by means of currents of the auditory nerve. Other achievements have been effected, and other researches are now being carried on. One of the most important things to be investigated is the problem of meningitis and other intracranial pyogenic diseases of otolaryngologic origin. Dr. Tobey suggests that during the course of the meeting, the society appoint a committee of leading otologists from each of the definite sections of the country who are ready or willing to organize a subcommittee in their respective centers, including one or two otologists, one or two competent neurologists, a neurologic surgeon, if available, and a neuropathologist who is interested or may become interested in this subject. He suggests that a comprehensive questionnaire be prepared and submitted and that the net results of this work be presented for discussion at each annual meeting of the American Otological Society. "This plan need in no way encroach upon the prerogatives of the individuals who are now carrying on investigations but would unquestionably be a means of saving many lives since it is the tendency at the present time to feel that something must be done although the why and the wherefore of what to do may not be known."

REPORT OF THE COMMITTEE ON PROGRESSIVE DEAFNESS. J. GORDON WILSON, Chicago.

Three broad lines of research were laid down by the bureau: (*a*) an anatomic investigation of the petrous temporal bone, (*b*) a genetic investigation of progressive deafness and (*c*) an experimental and metabolic (including endocrinologic) investigation of the production of progressive deafness.

This program was interpreted as allowing of flexibility; therefore a line of purely physiologic investigation has been added, namely, the physiologic investiga-

tion of conduction phenomena along the pathway of the eighth nerve. At present no funds are available to aid the last-mentioned research, but it is hoped that through the influence of the bureau, support will be forthcoming.

The genetic investigation was carried on by Davenport, and the results were recently published (Davenport, C. B.; Milles, B. L., and Frink, L. B.: The Genetic Factor in Otosclerosis, ARCH. OTOLARYNG. **17**:135 [Feb.] 1933).

In the anatomic investigation valuable contributions have been made under the auspices of the bureau. These include work by Bast and Anson on the development of the human temporal bone. Sections were studied, exact models were prepared and comparisons were made with various mammalian fetuses, resulting in a very complete study, now partly published.

The anatomy of the petrous bones has been studied with regard to the islands of cartilage, both in the fetal stages and after birth, and special attention has been paid to formation and postfetal changes. At present particular attention is being paid by both investigators to the fissula ante fenestram, especially in respect to (*a*) the theory much considered in Europe, that it is a product of synchrondrosis to relieve strains and (*b*) its association with otosclerotic areas which arise near or in connection with it. Working from different standpoints Bast and Anson have been able to correlate and check their findings. Papers are being prepared, and Bast has forwarded four models to the museum. Bast has also published an article on "Development of the Otic Capsule: I. Resorption of Cartilage of the Canal Portion of the Otic Capsule and Its Relation to the Growth of the Semicircular Canals" (ARCH. OTOLARYNG. **16**:19 [July] 1932). This has added to the knowledge of these primitive cavities.

Anson has made studies of the so-called islands of cartilage and their replacement by true bone. Their formation and disappearance in infancy and childhood have been discussed. He has also studied the cartilaginous otic capsule in various vertebrates, especially in the stapedial region.

The structure, form and location of otosclerotic patches in adult human ears were studied from microscopic sections and by means of a model prepared by sections.

Dr. Anson has also interested several graduate students in otologic research and with them has studied the comparative embryology and development of the endolymphatic duct with models and also the comparative embryology and development of the utricular valve.

Work on conditioned dogs has been carried on in the department of psychology at the University of Illinois and in the department of physiology at McGill University. Dr. Culler, of the University of Illinois, has tested the hearing of conditioned animals following plugging of the round window. He found that opening the bulla had no effect on hearing, but that a plug placed in the fossa of the round window impairs hearing, which is restored when the plug is withdrawn. He also found that such a plug reduces the electric impulses over the auditory nerve. This shows that electric impulses are related to, if not responsible for, hearing.

He found that firm pressure on the oval window may increase the electric pick-up from the nerve, but this is not constant.

Studies on the influence of x-rays on acuity of hearing in conditioned animals are still going on. So far the investigation has indicated an increase in acuity for some time.

Experiments on decorticated animals were difficult, owing to a high mortality. One animal was kept alive for some time and was rapidly conditioned to an electric bell. This shows that something corresponding to hearing was still present.

Dr. E. P. Fowler has carried on audiometric investigations at the Medical Center, and made routine examinations of temporal bones obtained at autopsy and from experimental animals fed on a diet low in calcium. By the ultraviolet microscope he has studied fibers of the petrous bone and their relations to cartilage islands and otosclerotic foci. With the aid of a revolving knife he has cut sections of undecalcified bone. He has also laid the foundations of an otologic museum.

Dr. Marvin Jones has established laboratories for pathologic studies at the Lying-In and New York Post-Graduate Hospitals. He has made a large collection of infant and fetal bones and has begun the microscopic study of these. He obtained funds for this work from interested persons.

REPORT FROM PROFESSOR TAIT'S LABORATORY AT MCGILL UNIVERSITY.

During the past year work on the hearing of animals has been carried on by Dr. Dworkin, Dr. Sutherland and Mr. Ross.

Dr. Dworkin reports that they have almost proved that in dogs the auditory region of the cortex is confined to the "Munk" area. The wider cortical expanse previously described appears to include areas for body tremor. In cats a new method of conditioning has been used in these experiments. Conditioned animals give the only valid test of the cortical registration of sounds. In these tests vibrating platforms have been constructed that operate in a soundless manner.

Dr. Sutherland has overcome many difficulties in experiments on conditioning. He has endeavored by these means to obtain a reliable audiogram for the dog. He has improved the generation of pure high pitched notes, and has utilized the sensitive flame as a measure of intensity.

Mr. Ross reports on the finer apparatus for detection of electric currents set up by the activity of the eighth nerve receptors. He shows that in fish otolithic receptors serve audition. He details his operative technic for ablation of the saccule or cochlea in cats, preparatory to the conditioning of these animals. Experienced persons who work on conditioned responses think that the real results are obtained from the unanesthetized, normal, moving subject. Reliable audiograms for each animal should be made before and after operative intervention. No conditioned test on animals has any value without carefully prepared audiograms.

Drs. McNally and Tait have worked exclusively on the equilibratory centers of the labyrinth, and later will work on the auditory receptors. They believe that the former provide important leads for work on the auditory receptors.

REPORT FROM THE CLARKE SCHOOL FOR THE DEAF. DR. NIELSON.

Studies on the auditory apparatus of deaf children have been made with a view to determining the character, location and extent of the defect. Studies also have been made on the immediate families of such children.

The results of two important studies have been charted. One chart represents the study of the descendants of one person who settled in America in 1630, together with a less detailed study of families related by marriage to the main family. Deafness seems to be on the increase in the descendants of this family, partly because many of them, both deaf and of normal hearing, married into families in which there was a long history of deafness or made consanguineous marriages and partly because of some environmental factor not yet discovered.

Another chart records deafness since 1700 in certain isolated families which settled in New England. Deafness reached its peak in this section in the middle of the nineteenth century and has been gradually disappearing, owing to the scattering of the families and to marriages with different strains. However, one of the persons in the Clarke School is a direct descendant of one family in which there were deaf persons in 1700, and another pupil is a descendant of this family through another line. In each case, one parent was from unrelated stock.

A study of auditory acuity for eight tones was made in seventy-five pupils, who were classified in four groups:

Group 1 was composed of six pupils with the largest amount of residual hearing. All heard loud speech, at least with one ear. With one exception all responded to the eight frequencies used. The hearing curve was higher at the ends. The response for the two ears at 512 double vibrations showed a loss of from 40 to 70 decibels. The response to similar tones heard by bone conduction was nearly normal for some and reduced for others, but the character of the curve was the same, with a dip in the middle. The vestibular response varied from normal to slight.

Group 2 comprised seventeen pupils with a small amount of residual hearing by air conduction. Many heard speech or appreciated its rhythm by means of amplifying devices. Of thirty-four ears tested 25 per cent showed a response to the highest tones. The curve had a descending character. There was a loss of from 40 to 95 decibels; at 512 double vibrations there was a loss of from 65 to 95 decibels. Tests of bone conduction showed a marked dropping off for high tones. The threshold for low tones was much lower than that in group 1. The vestibular response was negative in seven, normal in three and diminished in twenty-four.

In group 3 there were forty-one pupils with practically no usable residual hearing. The curve descended sharply, showing a marked loss of hearing for high tones. The vestibular response varied from none in ten persons to normal in sixteen.

Group 4 was composed of eleven pupils with the most profound type of deafness. Four gave no response to any sound on the *2A* audiometer. The remaining seven gave a response to only two tones at maximal intensity. The threshold for sounds heard by bone conduction was similar to that heard by air conduction.

OTOSCLEROSIS COMPLICATED BY OTHER LESIONS: A STUDY OF ROENTGENOGRAMS, AUDIOGRAMS AND LABORATORY AND CLINICAL FINDINGS. DR. EDMUND P. FOWLER, New York.

One hundred and twenty-six cases of otosclerosis collected over a period of six years are analyzed. All were carefully studied with the aid of the latest developments in otology, including audiograms, roentgenograms and differential and chemical examinations of the blood. With few exceptions, they had been diagnosed by other otologists as cases of otosclerosis. At autopsy, no doubt, some of these cases will show no otosclerosis, and many cases omitted from the list will reveal this condition.

Thirty-nine cases of otosclerosis were studied by microscopic sections in complete series and twelve cases by individual slides. The patients were divided into two main groups, those without complicating nerve deafness (eighty-one) and those with complicating nerve deafness (forty-five). In the whole series only 25 per cent showed no clearcut clinical evidence of previous inflammation in the middle ear. The patients in each of the two main groups were further classified as follows: (1) those without evidence of other complicating lesions; (2) those with evidence of previous masked or unrecognized inflammatory lesions in the middle ear; (3) those with a definite history of suppuration in the middle ear. A fourth group with existing suppuration was omitted so as not to confuse the picture.

Every patient suffered from bilateral deafness, and in most instances one ear was affected before the other. There was no instance of total deafness. Previous masked inflammation constituted fully 50 per cent of the complications found in the entire series. A history of progressive deafness appeared in from 50 to 70 per cent. A history of colds, exanthems and related conditions was very common. Tinnitus was present in a large percentage of the cases (from 82 to 88 per cent).

Dr. Fowler considers that except in the case of steady loss of hearing the variations in acuity were due more to variations in the pathologic process complicating the otosclerotic ankylosis than to any change in the bony ankylosis itself, and that any effectiveness of treatment was due to improvement in the condition of the middle ear and nasal spaces. He believes, however, that this is an approach to the therapeutic management of these cases which should not be despised.

A table showed the degree of involvement of the nasal sinuses in thirty persons with normal hearing, studied as controls. In these both the degree and the percentage of involvement were low as compared with the findings in patients with otosclerosis.

The almost universal evidence of previous inflammation in the nasal sinuses in the otosclerotic group and the comparative freedom from inflammatory signs in the controls still further strengthen the argument that inflammatory lesions of the middle ear are an etiologic factor in the deafness accompanying otosclerosis and, in fact, in otosclerosis itself.

Roentgenograms of the bones of the ear were of little if any significance, and examination of the blood, including the Wassermann test, indicated nothing of positive interest except that the otosclerotic process was present in spite of a normal blood chemistry. This does not mean that at the start of the otosclerotic process some change may not have been present.

Why does otosclerosis not appear more frequently if otitis media is prevalent and a probable factor in the etiology of otosclerosis? The answer is that it does appear more often but is not given as the diagnosis. Routine autopsies give an incidence for otosclerosis of more than 5 per cent of all persons, though few of these show bony ankylosis. This suggests that in routine autopsies on patients with deafness the incidence of otosclerosis is much higher. Pathologically otosclerosis is not a rarity; clinically it is a comparative rarity only because of failure of diagnosis. Photomicrographs from cases of otosclerosis were shown which demonstrated lesions with evidence of inflammation. The statements made were based on a study of five cases of otosclerosis from Dr. Fowler's collection in the department of pathology at Columbia University, fourteen cases of otosclerosis observed abroad and thirty-four cases of otosclerosis from the Johns Hopkins Hospital.

For controls he used his entire collection of about a hundred and sixty bones and a series of twenty consecutive cases studied by his son, Dr. E. P. Fowler, Jr., in the Johns Hopkins Hospital, through the courtesy of the research staff.

To summarize, in these cases of otosclerosis there was definite clinical evidence of the almost universal prevalence of postinflammatory lesions in the head spaces, particularly in the nose and the middle ear, and there were no abnormalities of the blood which could be linked with the otosclerotic process. The roentgen appearance of the sinuses strongly strengthens the clinical evidence. Syphilis may be excluded because the Wassermann reaction was positive in but a single instance. The pathologic evidence substantiates the clinical observations.

The terms progressive deafness and otosclerosis are not synonymous because other lesions often cause progressive deafness, and otosclerosis frequently causes no ankylosis of the stapes footplate and no deafness. The deafness accompanying otosclerosis may not be due wholly to bony ankylosis because there are usually complications which are capable of causing deafness.

Often the deafness accompanying otosclerosis is due wholly to a nervous lesion, and has no obstructive background. Examiners should keep in mind the fact that otosclerosis usually occurs coincidentally with other lesions of the ear. The detection of other lesions does not preclude a subsequent diagnosis of otosclerosis. The only therapy which promises prevention or arrest of otosclerosis is treatment to combat the tendency toward recurrent or chronic inflammatory processes in the nose and ear, particularly in the nasal sinuses, where the infection tends to linger before resuming an active state, with inflammation, suppuration, adhesions, thrombosis and ankylosis in the middle ear.

Especially is the otologist cautioned to search for otosclerosis even though his immediate diagnosis is definitely a residual inflammatory lesion of the ear, because otosclerosis is usually accompanied by evidence of such lesions in the past.

Treatment of the complications of otosclerosis is indicated not only as the only means of improving the hearing but as the only method which at present holds out any hope of preventing or arresting the otosclerotic process.

DISCUSSION

DR. THOMAS J. HARRIS, New York: This paper is broad and touches many sides of this important subject. There is no time to touch on all the points mentioned, so I shall confine myself to discussing the more important details. One of the strong points in this presentation is the emphasis on the relation of otosclerosis to disease of the middle ear. That is a thing all otologists are familiar with. Is it possible to make a differential diagnosis between the two conditions, or determine the etiologic relationship of one to the other? The question that I am particularly concerned with is whether or not one of our English confrères is wrong when he says that it is not possible to make a diagnosis of otosclerosis except post mortem.

I think that a number of points that Dr. Fowler makes are open to challenge. He makes an arbitrary distinction in his group of patients between those with and those without nerve deafness. He makes his premises without proofs, but these points do not bear on the larger question that he has brought up, namely, the question of disease of the middle ear. I think that he makes an original point when he shows the relation to previous disease in the nose and nasal sinuses. I differ from him in some of his methods of diagnosis. I think that the assertion that *all* cases of otosclerosis are bilateral requires confirmation that can be obtained only at autopsy. Clinically, I have seen cases in the beginning stages in which the condition occurred in only one ear. Dr. Fowler has voiced his condemnation of the Rinne test, and also stated his views concerning the low tones. There is considerable discussion on this matter, but he has gone further than most otologists in this respect in his substitution of the ratio between air and bone conduction. However much one values the audiometer, I think that it can confidently be asserted that some of Dr. Fowler's statements about bone conduction are open to question.

Although tinnitus is an outstanding and constant symptom in my experience, I question if one can say that it is pathognomonic of otosclerosis, in comparison with disease of the middle ear. Dr. Fowler says that otologists have failed to make a diagnosis of otosclerosis in many cases, and I agree with that.

Dr. Stacy R. Guild, Baltimore: When one thinks in terms of organic lesions otosclerosis is but one of the several distinct pathologic conditions which have been grouped together clinically as chronic progressive deafness. The only reason which has ever been seriously advanced for this grouping is an inability to make a differential diagnosis in a large number of cases. There is general agreement of opinion as to otosclerosis in typical cases, and almost as general agreement that when there is either a history or the signs of any form of chronic otitis media it is impossible with the present methods of clinical examination to say that an otosclerotic process is not also present. In microscopic sections the differential diagnosis is easy, and it may be that correlation of a sufficient number of complete clinical records with the histopathologic observations in the same cases will enable some one to establish a truly differential point clinically for otosclerosis. But a new method of testing for the presence of bony ankylosis of the footplate of the stapes appears to be needed, one which will distinguish an osseous from a fibrous tissue interference with ossicular movements.

As to Dr. Fowler's principal thesis, that inflammatory processes in the middle ear play an important rôle in initiating the otosclerotic process in the otic capsule, it seems to me, from the very nature of the facts that he has presented, that it is impossible to establish that the site of predilection is anterior to the oval window.

Might I ask Dr. Fowler how, on this basis, he would account for an otosclerotic area near the fundus of the internal auditory meatus, near the carotid canal or along the nonampullated ends of the vertical canals? The nature of the fibrous tissue in and near otosclerotic areas cannot well be regarded as evidence either for or against Dr. Fowler's theory, for its presence is easily and fully accounted for by the fact that the region is under the influence of the inflammatory process in the bone itself, otosclerosis being an osteitis in its essential features, whatever its etiology may be.

Dr. Emil Amberg, Detroit: In regard to the combination of otosclerosis with otitis media Politzer's textbook contains an important reference. Dr. Fowler divides his groups into (*a*) those showing no nerve deafness and (*b*) those displaying nerve deafness. One could distinguish further between patients with and patients without progressive labyrinthine deafness. Dr. Fowler said that there was no diminution of the calcium content. I should like to call attention to the admirable work by Bauer and Stein on the constitutional pathology of disease of the ear. They quoted Leicher's statement that there are two different kinds of calcium, one of which is the calcium ion. In pregnancy there is a lowered calcium content of the blood. There seems to be a definite relation between the calcium content in pregnancy and that in otosclerosis. In pregnancy the otosclerotic condition is aggravated. Another phase is the overstimulation of parathyroid function,

which diminishes the calcium content. As to the question of bilateral involvement, Bezold, I think, said that 88 per cent of cases were bilateral. Bauer and Stein thought that the effects on the ear are produced by effects on the general system. Heredity plays a great part.

DR. LOUIS K. GUGGENHEIM, St. Louis: In my experience also disease of the middle ear has been found in conjunction with otosclerosis but, I believe, as a mere coincidence. I should like Dr. Fowler to explain why one finds so many typical cases of otosclerosis when the tympanic cavity is free from any involvement. I should also like him to explain how involvement of the middle ear could bring about a bilaterally symmetrical otosclerotic process. Dr. Guild has mentioned otosclerotic foci far away from the tympanic cavity which could not be related to the tympanic process. We all look on Dr. Guild as an authority, and I was interested in his statement that an active inflammatory process is present in bone when otosclerosis exists. None of the specimens that I have examined have shown evidence of this inflammatory process. There are many cells present, including mesenchyme cells, fibroblasts, osteoblasts, histiocytes and osteoclasts, but I have never found round cells, leukocytic infiltration or any other sign of inflammation as I understand it.

DR. NORVAL H. PIERCE, Del Mar, Calif.: The diagnosis of uncomplicated fixation of the stapes can be made during life. With a normal drum head, patulous tube, increased bone conduction, loss of the limit for low tones to above 36 double vibrations, decreased air conduction and a history of heredity, the diagnosis is complete. One should not try to confuse this picture. Ankylosis of the stapes due to otosclerosis is sometimes combined with plastic processes in the middle ear. Such cases require the nicest discrimination in diagnosis. The diagnosis of plastic fixation of the stapes in a patient with an otosclerotic history presents still greater difficulties. Cases of otosclerosis in which the foci are situated elsewhere in the labyrinthine capsule than about the foot plate—that is, without ankylosis of the stapes—present the greatest diagnostic difficulties. Nevertheless, the occurrence of these types should not befog the fact that it is possible readily to diagnose cases of uncomplicated otosclerotic fixation of the stapes, these being by far the most numerous of this class.

In all histopathologic study of the ear one must remember that otosclerosis is not a disease of the periosteal or endosteal layers of the capsule but primarily of the endochondral layer.

DR. EDMUND P. FOWLER, New York: My object was to record the facts as I found them in the cases of otosclerosis studied and to point out what appeared to be some of the outstanding findings.

Dr. Harris asked, "Can you make a differential diagnosis?" I should say, "Probably as well as any one else can, by means of a careful and complete examination." I think that Dr. Harris misunderstood me when he said I claimed that all cases of otosclerosis must be bilateral. Observations at autopsy alone prove this is not true; nevertheless, the symptom of bilateral progressive deafness is an aid in the diagnosis of otosclerosis, and I have therefore mentioned it. I did not say that the Rinne test was of no value. I said that it was of limited value as compared with accurate audiometric tests by air conduction and by bone conduction, using either the audiometer or properly calibrated tuning forks.

I lay a good deal of stress on bone conduction because, although like air conduction it may be inaccurate, it enables one to attack the problem from another angle; without data on bone conduction over a spread of frequencies it is impossible, in my opinion, to arrive at as accurate a diagnosis as when the tests by bone conduction are made.

Had I used merely the calibrated tuning forks and not the audiometer in the tests, the audiograms in the different types of otosclerosis would have been just the same. There seems to be an idea that tuning forks and the audiometer give different results in hearing tests, but there are none if these instruments are properly calibrated and used.

Tinnitus is no more pathognomonic of otosclerosis than cough is pathognomonic of inflammation of the upper respiratory tract, but if constant, tinnitus, like a family history of otosclerosis, progressive and bilateral deafness, aids in arriving at a diagnosis of otosclerosis.

I agree with Dr. Guild that it is often impossible to tell whether the involvement of the middle ear found in microscopic sections is old or recent. Otosclerosis may begin years before any deafness is noted, and it may persist throughout the life of the patient without the presence of deafness.

Dr. Guild asked:, "If sclerosis depends on inflammation of the middle ear how do you account for its being present in various sites in the petrous bone?" I did not say that otosclerosis is necessarily due to disease of the middle ear as a whole or in part. I said that disease of the middle ear could not be ignored as a possible cause of otosclerosis. Even deep in the petrous tip one may see inflammation with no apparent connection with the middle ear and yet believe this to be the site from which it regularly originates.

Dr. Amberg recalls the fact that Politzer believed otosclerosis to be caused by catarrhal otitis media. I am not stating that it is caused by one kind of otitis media or another. I am simply recording different types of otitis media that I have found in otosclerosis and call attention to the practically universal occurrence of otitis media as proved clinically and pathologically.

As to the two kinds of calcium, I believe it is now claimed that there are several kinds of calcium in the blood. I have no report to make from this angle as yet.

I attempted metabolic estimations, but found tremendous variations and nothing which seemed to lead anywhere.

Dr. Guggenheim asked how I can explain the presence of otosclerosis without the indications of any inflammation. First, it is seldom that no signs of inflammation are present, and, second, signs of old inflammation are apt to fade out with the years and become increasingly difficult to detect. When I used the term "inflammation" I did not necessarily mean suppuration or even exudative inflammation. It must be remembered that there are various types of productive inflammation which are apt to leave nothing but a little scarring in the tissue attacked.

OTOSCLEROSIS, A REGRESSION? DR. LOUIS K. GUGGENHEIM, St. Louis.

The bilateral symmetrical involvement so frequently seen in otosclerosis suggests a gene-propelled dystrophy rather than a change in the tissues caused by venous stasis, irritation from the movement of the foot plate, pull on the tensor tympani, a spontaneous fracture or even a generalized metabolic disturbance, although these conditions may have an influence on the changes in the bone (for example, the appearance or increase of clinical manifestations during puberty, during and after pregancy, at the menopause, etc.).

In osteodystrophia fibrosa there is a generalized disturbance of metabolism which affects every part of the osseous system. Cases of osteogenesis imperfecta show a generalized metabolic disturbance affecting one of the smallest bones of the body and the densest, without change elsewhere in the skeletal system.

In certain lower vertebrates, for example, Clupea harengus (herring), the utriculus of one side is connected with that of the other by two canals, one passing above the brain and the other beneath, so that endolymph flows freely from one ear to the other. There exists, as it were, a single ear. When the otosclerotic ear is regarded as a lower vertebrate structure in the sense that lower vertebrate genes are present and certain lower vertebrate forms develop, it is easier to understand the bilateral symmetrical involvement of the "one ear" than such a change in two entirely separate structures.

When in otosclerosis the foot plate and annular ligament fuse with the capsule (return to the tissues whence they were derived) a capsule is formed which suggests that of the lowest vertebrates.

Myxine glutinosa (hagfish), possessing but two canals and a vestibulum, has a cartilaginous capsule entirely devoid of external openings such as oval and

round windows. Petromyzon fluviatilis (river lamprey) shows the addition of a sacculus but possesses a cartilaginous capsule with no windows. In Acipenser sturio (sturgeon) one notes the addition of a third canal and a lagena cochleae but no windows in the capsule.

The cochlea and the two windows are recently acquired structures, and I agree with Gray that otosclerosis represents a variation in these recently acquired structures. I believe, however, that the variation is not an accidental one but rather the result of a regression manifesting itself after the normal genes have expressed themselves. Most frequently the oval window, first to appear, shows an attack by the genes of regression. Not infrequently, however, the cochlea and round window are involved. Rarely does one see involvement of the region about the canal; this involvement, conceivably, may result from an attack on the third canal not present in the lowest vertebrates. In other words, the genes of the lowest vertebrates, in expressing themselves, tend to obliterate such structures as do not belong to the type of capsule that they are destined to build.

The mammalian ear, in its ontogenesis, passes through the various stages of phylogenesis. When the auditory placode invaginates and is cut off from the ectoderm, there results in the auditory vesicle an aural organ similar to that in the lowest forms of life, for example, in Ascidia. The mesenchyme surrounding the vesicle condenses and then differentiates from the remaining cephalic mesenchyme. No windows are present. At 2 months (human age) mesenchyme is converted into perichondrium and precartilage. Two weeks later the capsule is of cartilage except where the oval and round windows will develop; in these two areas a mesenchymal curtain stretches across. The annular ligament begins to differentiate. The stapedial ring from Reichert's cartilage, which will form the crura, is seen in close relation to the curtain. Under higher magnification one sees the mass of mesenchymal cells of the curtain and the cartilaginous ring. The curtain, derived from the capsule, will form the foot plate. The annular ligament is beginning to differentiate. At 3 months (human age) the capsule of mammals shows full development of hyaline cartilage such as exists in the lowest vertebrates. At this period the stapedial ring has flattened medially to accommodate itself to the curtain (now of precartilage). The annular ligament is differentiating. At 4 months of age the ring has fused with the cartilaginous curtain which may now be designated as the foot plate of the stapes. The foot plate cartilage is younger than the ring; therefore it stains less deeply. The annular ligament is fully differentiated. Under high magnification one sees pluripotential undifferentiated mesenchymal cells on each side of the annular ligament. These cells are of great importance in the development of otosclerosis. At this period the stapes has attained the adult form. Ossification of the capsule is well under way. At 4½ months ossification of the capsule is progressing rapidly, but stops abruptly on either side of the oval window, where cartilage persists. The difference in texture between the crura and foot plate is hardly discernible. The curtain over the round window has now differentiated into the membrana tympani secundaria.

In fishes the oval window does not develop beyond the membrane-covered opening. In Proteus anguinas (first order of amphibians) the oval window is closed for the first time by a bony operculum. No annular ligament or ossicle is present. A pars initialis cochleae with a membrana tectoria has appeared. In Menopoma alleghaniense (hellbender) one finds the first ossicle with a foot plate articulating with the oval window by means of an annular ligament. In Rana esculenta (edible frog) one finds an ossicle (columella) of bone and cartilage with a foot plate articulating with the oval window. A round window covered with membrane makes its appearance. Most interesting of all is the presence of the canalis fenestrae ovalis, a passage-way from the perilymphatic spaces, which passes through the external capsule wall between the fovea fenestrae ovalis and the foot plate to end as a blind sac in the tympanum. This canal, originating with the utricular projection of the shark and ray stages, has now severed its connection with the endolymphatic spaces and approaches more nearly the form of the fissula ante fenestram of man. Evidently in amphibians and higher vertebrates, the accessory endolymphatic duct

is no longer needed. Thus one may trace the origin of the oval window and the fissula ante fenestram, both of which are important in the development of otosclerosis.

The site of predilection in otosclerosis is anterior to the oval window, the position of the fissula ante fenestram. This structure, which passes from cisterna to tympanum, is present in all human embryos and sometimes persists throughout life. At 13 months of age the fissula is a well developed passage-way just anterior to the foot plate. An early focus of otosclerosis developing in the region of the fissula is demonstrated. On higher magnification of the normal fissula one notes a heavy cartilage wall, and under oil immersion the canal is seen to contain numerous pluripotential undifferentiated mesenchymal cells and other elements. When these mesenchymal cells contain the genes of lower vertebrates, otosclerosis may develop. When they are inhibited sufficiently to contain normal genes or when irritation is lacking otosclerosis may not develop but may be transmitted.

The fissula post fenestram, an indentation of the capsule posterior to the oval window, is entirely a mystery. Bast thinks that it may be a drainage point for the perilymph. Under higher magnification the fissula in man, at 4½ months of age, is seen to be lined with fibroblasts, some of which are invading the cartilage of the capsule.

The marrow spaces of normal as well as of otosclerotic capsules contain pluripotential undifferentiated mesenchymal cells which under oil immersion are well visualized near a haversian canal. Some marrow spaces are swarming with these cells.

The pathogenesis of otosclerosis may be briefly described as follows: In the fissula and marrow spaces mesenchymal cells containing the genes of regression proliferate into a mesenchymal sponge. Pressure is exerted. Cartilage cells succumb. Differentiation of the mesenchyme occurs into fibroblasts, histiocytes, osteoblasts, new vessels and sometimes osteoclasts. The adjoining capsular bone shows preparedness, which is identified by poor staining and the absence of many details noted in the normal bone. This preparedness probably results from pressure, interference with circulation and possibly also from the chemical action of the products of degeneration of cartilage. New vessels from the otosclerotic focus penetrate the prepared capsular bone. Resorption follows. The blue-mantled vessels are surrounded by cementum. The so-called giant cell osteoclasts sometimes seen in areas of resorption are more probably phagocytes, as the same process of resorption occurs in cases in which these cells are not found. Pink-stained osteoid tissue is sometimes seen. Osteoblasts in epithelial alinement may be seen, but true lamellar bone does not, as a rule, form. The penetrating, newly formed blood vessels find themselves surrounded by many mesenchymal cells, histiocytes, fibroblasts and osteoblasts. The fibroblasts may lay down connective tissue at the periphery of the resorption spaces. Soon calcium is deposited, and *Geflechtartig knochen* (irregular fiber-bone, braided bone, calcified connective tissue or otosclerotic bone) is formed. The areas of resorption, containing vessels surrounded by cellular elements, give rise to the porosity of otosclerotic bone.

Studying the region of the annular ligament posteriorly and anteriorly, one notes a plate of cartilage over the foot plate and over the capsule. This tissue is sensitive to mesenchymal attack. In typical otosclerosis, the focus anterior to the foot plate sends out myriads of mesenchymal cells (seen swarming through the ligament toward the foot plate). An area of resorption in the foot plate is seen; the foot plate is being transformed into braided bone. Soon the ligament will be obliterated by a deposit of calcium; the capsule is closed and thus regresses to its lower vertebrate form.

As time goes on more and more fiber-bone is deposited; the focus becomes denser and denser. The resorption spaces lose their rich cellular content and show instead fibrous tissue. The blood vessels become smaller, and many disappear.

In conclusion, an objection to the theory of regression may be stated: In man no other instance has been found in which a tissue reaches normal development and then shows regression to a lower vertebrate structure. There are many

vestigial structures in the human embryo, some of which disappear before birth while others persist throughout life. Examples are the vomeronasal organ of Jacobson, notochord, fissula ante fenestram and branchial clefts. Fischer, of Vienna, reported a case in an adult with a third macula, reptilian cristae and other lower vertebrate structures. Ruttin reported a case of a macula neglecta. The only example of regression in a normally developed tissue is found in a low form of life, Ascidia. This organism, when fully developed, moves about freely, possesses a brain vesicle, an aural vesicle with otolith and special organs for tactile sensation and perception of light. Within a very short time it regularly loses its brain vesicle and all its sense organs and regresses to a sessile organism.

DISCUSSION

DR. J. GORDON WILSON, Chicago: The statements that in otosclerosis the changes in the bone are confined to the aural labyrinth, that the foci are bilaterally symmetrical and that there is a site of predilection are generally recognized as true and call for no discussion. It is otherwise with the suggestion that otosclerosis is a regression and the theory expressed regarding the origin of the foot plate of the stapes.

In support of the former theory it is suggested that the fissula is a vestigial organ corresponding to a structure found in certain fish. Dr. Guggenheim is not the first to be puzzled by the fissula and to seek for an ancestor. Both Bast and Anson were equally puzzled and sought for it in various mammals or for a trace of it in the fetal stages of various mammals. But so far no trace has been found except in man. Since no corresponding structure to the fissula ante fenestram can be found in birds or in mammals except man, it is a risky procedure to point to a structure in a low vertebrate form as the predecessor or homolog of the fissula without explaining what happened to it or why it disappeared during the million odd years which intervened between the appearance of fish and the advent of man. Attempts to class as homologs structures which superficially look alike or occupy the same site in fish and in man, with no corresponding structure in intermediate vertebrate divisions, has led many morphologists astray—for instance, those who have insisted that the teeth of the shark and the teeth of man are homologous structures. The chromosome hypothesis to which Dr. Guggenheim refers is, as he knows, a working hypothesis which may or may not be true and cannot be taken as a basis for such a deduction as he makes.

I am not convinced that the foot plate of the stapes is derived from the otic capsule. It certainly does not appear to be so in the human fetus. In very early human embryos the primitive cartilage from which the foot plate is derived comes from the second branchial arch. In very early embryos, just as cartilage is appearing, it can be seen lying in the fossa ovalis, separated all around from the wall of the fossa by a surrounding mass of mesenchymal tissue.

DR. EMIL AMBERG, Detroit: Imagination is not always a safe guide, but one must sometimes construct a hypothesis in order to work out a particular result. Osteitis fibrosa is a lesion that is entirely distinct from otosclerosis, in my opinion. The parathyroid glands have a special bearing on calcium metabolism and perhaps on otosclerosis. Brunner and Stein showed a large number of degenerative changes in connection with otosclerosis. The mechanism of the disease is very important. Ballin, of Detroit, is doing excellent work on the parathyroid gland. Szasz of Budapest published an account of a case of acromegaly with otosclerosis in which hearing was improved by roentgen therapy directed to the hypophysis. There are two theories on which this work is based. One attributes the improvement in hearing to the influence of irradiation on the hypophysis; the other theory, that of the Spanish school, attributes it to the effect on the sympathetic nervous system. Acromegaly has a direct relation to hardness of hearing, but there seems to be a distinction between the effects of acromegaly and those of parathyroidism. In regard to genetics, Hanhart followed the history of a Swiss family for three hundred years, and found that one couple was responsible for all the cases of

deafness that appeared in descendants during that period. Recessive qualities were dormant, and intermarriages produced deaf children. Dr. Guggenheim spoke of changes in the mechanism of hearing. How did they first come about? The sins of the fathers are apparently visited on the children "to the tenth generation." Consanguineous marriages have some influence on recessive qualities. The influence of the father and mother may perhaps not cause changes in the next generation, but in the following generations an influence may be felt, for example, in cases of color blindness. Professor Little has shown in his work on mice that the immediately succeeding generation is not affected, but the next one is. Another point is that there seems to be some positive relation between otosclerosis and carcinoma, but a negative relation between otosclerosis and tuberculosis. It is claimed that members of families with otosclerosis are long-lived. The longer a person lives, the more likely is he to become a victim of cancer. That point must also be considered. It is not only in the ear that recessive tendencies are shown. For instance, in incest, the offspring have been shown to suffer from ichthyosis.

Dr. Alfred Lewy, Chicago: A few years ago, I read a paper before the Triological Society, describing the experimental feeding of calcium fluoride to white mice. It had long been known that fluorine produces changes in bone, and this experiment was conducted to determine whether the bony labyrinth was also affected. In some of the experimental animals the bone appeared less mature than in the controls. Further study showed in one specimen the deposition of osteoid tissue on one of the crurae of the stapes and on the labyrinthine wall nearby. This evidence of the effect of fluorine is somewhat clouded by the presence of otitis media, which occurred in all of the experimental mice and is difficult to prevent. However, the experiment suggests a line of biochemical approach in the study of otosclerosis.

Dr. Moŝes H. Lurie, Boston: What is the relation of the oval window in the rajah to the first pharyngeal pouch? Does Dr. Guggenheim refer to the projection from the ductus endolymphaticus of the animals?

Dr. Stacy R. Guild, Baltimore: In regard to Dr. Guggenheim's statement that he has never seen evidence of an inflammatory process in otosclerotic areas, I should say that in the so-called "active" stage the process frequently has a histologic appearance which so resembles in some respects that of osteitis that it seems entirely possible that much fibrous tissue may be produced by the reaction. I shall be glad to show him such specimens.

Dr. Louis K. Guggenheim, St. Louis: Dr. Wilson is right. This paper may even be stamped as pseudoscientific. However, sometimes truth can be arrived at in this way. One cannot be certain that the accessory endolymph duct in the shark and the ray is the forerunner of the fissula ante fenestram, but it makes interesting speculation. The fact that the fissula ante fenestram is not present in the intervening stages of evolution is interesting, but it does not prove that the genes may not have been merely inhibited, and are therefore capable of later activity. I am not quite clear as to Dr. Wilson's comment about the origin of the foot plate of the stapes. I think that it is derived from the capsule and not from the second arch.

Dr. J. Gordon Wilson, Chicago: I examined a 6 months fetus and an earlier one, and in the latter the cartilage was not developed; it was in an earlier stage between mesenchyme and cartilage. I think that it is derived in the manner described by embryologists, but I should like to believe that Dr. Guggenheim is right. It would help one a great deal, but more evidence is needed.

Dr. Louis K. Guggenheim, St. Louis: The human specimens that I have examined do not show details as clearly as the specimens from rats. In the latter the foot plate is clearly derived from the capsule; the crura, from the second arch. I find that at 4 months of age, although the foot plate and Reichert's ring have fused, the two are different structurally. Gradenigo stated in 1879 that the foot plate was not derived from Reichert's cartilage. As for the fissula, Bast thinks that it may be vestigial. I know that there is a lack of certainty in my statements. The work is not conclusive, but many of the scientific facts of oto-

sclerosis fit in with the theory of regression. Dr. Amberg misunderstood my statement about osteodystrophia fibrosa. In such conditions the entire osseous system may be involved, while in otosclerosis the foci are confined to the labyrinth. Dr. Amberg asked about genetics. I know too little about genetics to discuss the subject, but I try to follow Davenport who says that in otosclerosis there are two dominants, one sex-linked. Dr. Amberg said that otosclerosis may skip certain generations, and appear at a later period. This is, of course, quite true. In the present theory genes of regression may be inhibited only or by permanently stilled. We obtained recently by cesarean section a human embryo of 2 months in a classic case of otosclerosis. We are hoping that this specimen will show very early evidence of otosclerosis, at the precartilaginous stage, that is, certain abnormalities of the differentiated mesenchymal cells. Dr. Lurie asked if the first pharyngeal pouch of the ray is in direct relation to the oval window.

Dr. Moses H. Lurie, Boston: The relation should be the same in man as in the lower animals.

Dr. Louis K. Guggenheim, St. Louis: The first pouch forms the eustachian tube, but there is no direct relationship between the first pharyngeal pouch and the oval window, except that the oval window abuts on the lateral expansion of the pouch (the tympanum).

Dr. Moses H. Lurie, Boston: The endolymphatic duct in the rajah opens on the skin just laterally to the spine and fills the membranous labyrinth with sea water. I do not know whether you called that the oval window or whether you mentioned another aperture. That is the first evidence of the formation of the oval window. Is it related to the first pharyngeal pouch?

Dr. Louis K. Guggenheim, St. Louis: The first sign of development of an oval window is in the herring. It is in the wall of the capsule and is not to be confused with the opening of the endolymphatic duct. Dr. Lewy's experiments with fluorine are interesting.

Effects of Drugs on Vestibular Reactions. Dr. Ellison L. Ross, Chicago.

This article will appear in full in a later issue of the Archives.

Afternoon Session, May 8, 1933

Interpretation of Audiograms. Dr. Stacy R. Guild, Baltimore.

The growing tendency on the part of many otologists to make certain diagnoses almost wholly on the basis of audiograms is, in my opinion, not justified by the facts. The procedure reflects rather the hope which many of us had when audiometers of modern type first began to be used, namely, that for each pathologic lesion of the ear there would prove to be a characteristic audiogram. No one believes more firmly than I do that acuity for tones by air conduction can be more accurately determined by the careful use of a well calibrated audiometer than by any other means now available. But even the best of audiograms are records of acuity by air conduction only. Complete examinations by every available method are just as necessary as ever. The importance of the classic tuning fork tests in otologic diagnosis is not decreased by the use of an audiometer.

The numerous cases presented by means of lantern slides (audiograms, other functional and clinical data and histologic sections) are arranged in a sequence which emphasizes the differences which the other data may show when the audiograms are similar. The difficulties of differentiating, by means of the functional data, between true otosclerosis and certain other conductive lesions are stressed. Patients whose audiograms indicate that all high tones are poorly or not heard, while the low tones are well heard, have shown the most consistent pathologic changes of any group thus far studied. The essential lesions are in the basal turn

of the cochlea, and consist of partial or complete atrophy of portions of the organ of Corti, the nerve fibers in the osseous spiral lamina, the cells of the spiral ganglion or all of these elements.

The necessity of adequate "masking" of one ear in tests of the other one is not as generally recognized as it should be. Some of the cases presented stress the importance of this point in the technic of clinical examination for both air and bone conduction.

DISCUSSION

Dr. Edmund P. Fowler, New York: I think that there is no question but that we all agree with Dr. Guild that one cannot make a diagnosis on the basis of audiometric tests alone, and that to get an accurate picture of the condition in both ears one must use a masking tone to see if there is a great difference between the two in acuity of hearing. The great difficulty in the use of masking sound is that when the hearing is greatly lowered the masking sound cannot be made to enter the ear in sufficient volume. How much noise the ear requires for masking is not known unless the sound is first used on both ears to test the efficiency of the masking and then used on one ear alone. I think that the Bárány "noise" apparatus makes too much noise in the room; it really masks the hearing of both ears when used in one. The best way is to produce the masking tone within the auditory meatus, near the drum membrane, by means of an air current blown into the external auditory meatus through a small nozzle. One must, of course, weigh the family history in connection with the otoscopic examination, giving due significance to the course of the disease, the flush of the promontory and other factors.

One should test for bone conduction by tuning forks or by audiometer even if the tests are not exactly accurate. My experience is that bone conduction is seldom diminished more than air conduction. In Dr. Guild's charts, the curve is down so far that one cannot get bone conduction by forks or by audiometer because neither produces sufficient loudness of sound. The limits for intensity of sound in bone conduction range about the 40 or 50 line with the *2 A* audiometer. Above 4,000 double vibrations bone conduction is very inaccurate. A comparison of air conduction with bone conduction by tuning forks is misleading, because one usually obtains no ratio between the two. If, however, the method that I advocated several years ago is used, namely, the alternate placement of the fork, first on the mastoid and then before the ear, for two and a half seconds, the decrement of the fork is the same for bone as for air. It dies down, in other words, in very small steps but in an even manner for both air and bone conduction.

Regarding a step-up result in inflation of the ears because of aeration of the middle ear, I am rather skeptical. In my opinion, if the eustachian tube is really closed the drum becomes plastered against the promontory. The majority of otosclerotic patients have overinflated middle ears, owing to overtreatment. In the Hudson tubes, in normal persons, there is a sense of pressure, usually relieved by swallowing, but some persons have to blow the nose hard to relieve the sensation and reestablish patency of the eustachian tube. Often the reason that a tube does not appear patent is that the swallowing is not coordinated with an increase in air pressure.

In bone conduction tests with the forks, the fork should not be pressed too hard on the skull because this causes strong damping of the forks. With reference to cases of perforation of the drum, every otologist has seen patients with no drum but with almost normal hearing. Often an ear with chronic suppurative otitis hears better than the other ear, in which the perforation has healed; after one has performed a radical mastoid operation and cleared up the suppuration the hearing may be worse. If the suppuration originates in the eustachian tube it is safe to say that the patient will die of something else, but if it arises in the attic it is a real danger. Therefore one must determine where the chronic suppuration is located. One should try to cure the suppuration in the attic and mastoid, but even if it is in the latter it is not always necessary to perform a radical operation. Whether a skin graft is used or not, the hearing depends on the amount of scar tissue that remains. The main point that Dr. Guild notes is correct, that

one cannot make a diagnosis from the audiometer alone. It is obvious that any obstruction in the middle ear has an action depending on its location. The most important area is the foot plate of the stapes. Deafness in otosclerosis is caused not only by ankylosis of the foot plate to the margin of the oval window but frequently by adhesions around the foot plate of the stapes as well.

Dr. Samuel J. Crowe, Baltimore: I think a great amount of information can come from experimental and pathologic work, particularly when an effort is made to correlate it with clinical findings. Of course the experimental results obtained by testing animals with the Wever and Bray method cannot be interpreted literally to explain the defects in hearing observed in patients, and to a less extent this applies to the study of histologic sections of the human ear. Both of these methods, however, furnish accurate data on which to base an opinion. The entire staff of the Otological Research Laboratory in Baltimore has been engaged during the past three years in an effort to find a rational anatomic and pathologic interpretation of audiometer and tuning fork tests made on patients with the commonest of all hearing defects, a loss or impairment of perception of high tones. Dr. Guild mentioned these studies briefly, since the main object of his paper was to direct the attention of the clinical otologist to the fact that an audiometer test alone is entirely inadequate. A careful and detailed clinical history and examination of the tympanic membrane, eustachian tube, upper air passages, cranial nerves and general physical condition are necessary, but a detailed tuning fork test is absolutely essential. The audiometer record supplements but in no way replaces the older, well established methods of examination.

The main object of our research laboratory is to try and correlate experimental findings, audiometer records, tuning fork tests and clinical data on a large group of patients and thus furnish the otologist with anatomic and pathologic facts on which to base the interpretation of his clinical tests.

Dr. Robert Sonnenschein, Chicago: Laboratory work must be correlated with the clinical findings by postmortem study wherever possible. Bezold did all his splendid work by means of clinical examination with tuning forks and voice tests, together with postmortem examination whenever possible. Audiometers have many advantages, but they are not indispensable for the diagnosis in otologic cases. In testing bone conduction I consider the fork more reliable than the audiometer. For the highest tones, the monochord is most reliable for both air and bone conduction. To eliminate the objections that are usually raised against the Rinne test, Minton and I modified this test a good many years ago so that the stem of the fork could be used for both air and bone conduction. It is important in speaking of the Rinne test to state not merely whether it is negative or positive, but to what extent air conduction is longer than bone conduction or vice versa. Neumann's electrical noise apparatus is better than the Bárány apparatus, as it does not produce as much extraneous noise.

Dr. C. C. Bunch, St. Louis: I agree with Dr. Guild in the statement that one cannot make a diagnosis on the basis of the audiogram alone. Complete and thorough clinical examination is always essential. Those who are constantly using the audiometer find errors in technic which often account for peculiar and incomprehensible results. Simple matters such as the proper adjustment of the telephone receiver at the ears, the proper tension of the head band if one is used and the conditions of noise in the room where tests are made play an important part in securing accurate results. These factors are especially important when one is interested in making repeated tests on the same patient to determine the progress of deafness if treatment is undertaken.

Dr. Louis K. Guggenheim, St. Louis: The end-organ is not the whole story. In the medulla, at the level of the inferior olives, are the cochlear nerves, anterior nuclei, cochlearis, tubercula acustica and other structures. From the tuberculum pass the striae acusticae, etc. I should like to ask whether the workers at Johns Hopkins have studied these intracranial structures along with the end-organs, when

the audiograms cannot be interpreted, to see if the difficulty may not be explained by changes in the central structures?

DR. HORACE NEWHART, Minneapolis: Audiograms have now reached the laity. The other day I was asked whether an aging teacher should be asked to resign on account of her audiogram. The otologists here may be taxed with a similar question, and I think one should give but one answer in such a case; that the status of a teacher should be based on her record and her performance, and not on an audiogram or results of tuning fork tests.

DR. EMIL AMBERG, Detroit: It has been my experience with school children that after the audiogram is obtained the ear is sometimes found to be obstructed by wax. Of course, the wax is removed, and another audiometer test requested. This is a small but important point. I should like to call attention to an alleged racket that is going on in this part of the country in connection with the aurotone or oratone. Dr. Shambaugh examined the results obtained with this machine. His report (The Oratone, Correspondence, *J. A. M. A.* **99**:1102 [Sept.] 1932) should be read carefully. It is distinctly the duty of physicinas to look into the matter and to advise patients according to the merit or lack of merit of this apparatus. It has a wonderful psychologic effect, and I can well understand its general influence when the patient's nerves are on edge.

DR. DOUGLAS MACFARLAN, Philadelphia: In quantitative tests I make use of the tuning fork. I use a standardized blow, a swinging pleximeter. Timing the fork gives the best knowledge of the contrast between air and bone conduction and is valuable in making a differential diagnosis. I think the "on and off" method with the tuning fork is the best one for obtaining the distinction between air and bone conduction. With it the examiner can use one blow and let it die out evenly, and the patjent gets the contrast between sound and the absence of sound. This method is important for another reason: Deafened patients are likely to get the impression of a tone image with the single tone if they are exposed continuously to it.

I am sorry that Dr. Guild did not have time to speak about the phonographic audiometer which takes the place of the old spoken voice test. The phonograph gives one an idea of central perception which is a complicated affair, dependent to a large degree on educational and psychologic factors. There is no correlation between the hearing of single tones and of speech. In speech one is listening to a very complex thing.

On all of the records there should be notes as to the "set" of the test and as to the type of patient. An idea should be given of the sort of person one is dealing with, whether he is alert or dull, intelligent or illiterate. If he is a child, is he eager, timid, cooperative, resistant, indecisive, etc.? These factors are important.

DR. EDMUND P. FOWLER, New York: Dr. Guild says that a certain type of curve is associated with ganglionic disturbance, but if one finds normal bone conduction I cannot see how there can be much disturbance of the nerves. Bone conduction is the greatest aid in determining nerve deafness and yet Dr. Guild does not use it. Dr. Sonnenschein misunderstood what I said about tuning forks. The audiometer as it is now used is more reliable than the tuning fork. When one uses the fork in the customary way for air and bone conduction one might as well use two instruments, one of which gives a response for so many seconds and the other a much smaller response, which dies down much faster because of pressure on the head.

DR. STACY R. GUILD, Baltimore: From the amount of discussion it is evident that there are many others who have difficulty in interpreting the results of functional otologic examinations. I did not mean to give the impression that I am discouraged but merely that a great deal more work remains to be done. The more complete the clinical examination is, the better are the chances for final success in correlating the several items and the whole picture with the underlying forms of pathologic change.

TACTILE SENSATION AS RELATED TO TESTS OF HEARING AND HEARING IMPRESSIONS THROUGH OTHER NERVES THAN THE EIGHTH. DR. DOUGLAS MACFARLAN, Philadelphia.

The response to the feeling of the vibrations from testing instruments used to produce sound frequently deceives the examiner into thinking that the patient is reporting sound. Many profoundly deaf persons, especially children, have little or no concept of sound. One must then be sure of what these persons are reporting. This has necessitated a more thorough understanding of the normal tactile response about the ear.

Vibrations between 128 and 2,000 may be felt if delivered in strong enough intensities. Deaf persons apparently show a greater tactile acuity than do normal persons, for the reason that in the latter the hearing of the loud sounds that these instruments produce overshadows the first sensation of feeling the vibrations.

A convenient test for differentiating between feeling and hearing is one described by the author. A sounding tuning fork is held vertically with one edge facing the external auditory meatus. The patient is directed to report by raising his hand when sensation is noticed. The hand is to be dropped when the sensation is gone. Both the greatest sound and the greatest tactile stimulus are coming from the flat of the tine which is facing the pinna. Tactile sense should disappear first, as it is less acute and lessens rapidly with the dying down of the fork. When the patient's hand is dropped, the fork is rotated so that the flat of the tine faces the meatus. If the hand is again raised, the patient is reporting hearing.

(Graphs were shown illustrating the relative acuity of tactile sense about the ear as compared with that in other areas of the body. The amount of overlapping of the two senses about the ear was illustrated.)

A case is reported in which hearing could be achieved by means of greatly amplified sound. Various pitches of great loudness presented to the ear could be imitated by the patient. Sound delivered by a loud speaker unit, minus the cone, was perceived as sound by the finger-tips. The same sound was not sufficiently strong to have been air-borne to the ear. The phenomenon was not bone conduction, as the effect was produced with the receiver not on the olecranon but on the ulnar nerve in its groove, a fraction of an inch from the olecranon. The patient could match exactly the pitches perceived in this manner, and could recognize the difference between phonographic speech, intrumental music and singing sent out through an electric pick-up.

The only explanation ventured is that crude tactile sense occasionally exhibits the rudiments and perception of the sound frequencies which are so well perceived by the special organ of sense, the ear, which develops from the crude tactile sense.

DISCUSSION

DR. C. C. BUNCH, St. Louis: Dr. Macfarlan is to be congratulated on stepping aside from the usual routine and pointing a way to a line of study which may have distinct clinical and practical value. From the evidence presented it must be granted that his conclusions are in opposition to the doctrine of specific nerve energy. There can be no doubt that his patient heard the stimulating tone, since she was able to reproduce its pitch vocally. This requisite for agreement with the doctrine of specific nerve energy is evident. One may also assume that the ulnar nerve might conduct this specific nerve impulse. There is, however, no evidence of the presence of the third requisite, namely, a special receptive organ at or near the point of contact of the bone conduction receiver and the nerve, to receive the mechanical stimulation and transform it into a nervous impulse.

To check the results presented, I conducted tests on two persons with normal hearing, both of whom were skilled psychologic observers. With a Western Electric audiometer and a bone conduction receiver similar to that described by Dr. Macfarlan, tests were made to see if these observers could "hear" the tones when the receiver was pressed against different parts of the body. To eliminate the possibility of these listeners hearing the tones by direct air conduction, their

ears were forcibly closed by inserting the tips of the forefingers into the external canals. This proved sufficient to prevent the entrance of the sounds through the canals by air conduction. The receiver was then held against the body. When the receiver first touched the clothing of the body, the part in contact was thrown into vibration and, as might be expected, the sounds became louder in the room but not sufficiently loud to be audible to the two listeners. With increased pressure the tones were audible at many points of contact on the body. They were heard most distinctly when the receiver was pressed against the spine, the large muscles around the shoulders, the ribs, the abdomen and even the patella, when the listener was seated. The sound was less distinct with the receiver pressed against the soft parts of the body than with it pressed over the bones.

It is true that these experiments do not duplicate the conditions reported by Dr. Macfarlan. But the conclusion reached was that sounds reached the ears through the continuity of tissue. In Dr. Macfarlan's case there was the same continuity of tissue, so the conditions were similar except that my listeners were not deaf. I am, however, at a loss to understand, on the basis of my experiments, why his patient heard when the receiver was over the ulnar nerve (a softer part of the body) or on the finger-tips and not when it was on the olecranon process.

DR. ROBERT SONNENSCHEIN, Chicago: In a splendid address made by Dr. Max A. Goldstein, president of this society in 1928, he said: "The sense of touch seems to be the most generally distributed, and from this sense, the other senses seem to have been in most cases developed. In the lower order of animals, the special sense organs are not usually sharp enough to distinguish one from the other, and in some instances, the eminent naturalists who have engaged in this delicate research conclude that the same nerve is capable of carrying different sensations, according to the structure of the end-organs." After discussing in his address tactile and other senses in insects and other forms of life, Dr. Goldstein further says: "The practical application of these anatomical and physiological principles has found a significant outlet in the various forms of intensive training of the handicapped child, having for its prime object increased stimulation of the organs of special senses, either individually or collectively."

As a matter of fact, this is well exemplified in the work of Prof. Robert H. Gault, with his "teletactor." In a report of some of his results, in 1926, he showed that by the tactile sense of the fingers certain persons learned to detect not only rhythm but actual words and sentences. Long ago, Dr. Goldstein, in his training of deaf children, used a megaphone with a diaphragm over the large end, against which the fingers of the children could be placed at the time of instruction.

Dr. Macfarlan ingeniously devised the tuning fork test to distinguish between the tactile and the hearing sense. It is quite true that the angle of the prongs affords the least intensity of sound. May I be permitted to make a small technical criticism of the statement that both the greatest sound and the greatest tactile stimulus come from the flat surface of the tine or prong? In a paper read before this society in 1924, "A Study of the Intensity of Sound from Various Parts of the Tuning Fork Prongs," I stated that after careful experiments with electrically driven and otherwise activated forks, by means of the Webster phonometer, it was determined that the outer or flat surface of the prongs gave forth the greatest intensity of sound, and that the two narrow surfaces held parallel with the ear gave the next greatest intensity. I corrected this statement, however, after work done with Mr. Eisenhour at Riverbank Laboratory. I presented the results before the Middle Section of the American Laryngological, Rhinological and Otological Society in January, 1929. The shape of the fork and the distance between the prongs are of considerable importance. In other words, in certain forks the greatest intensity is given off from the outer surface of the prongs, and in others from the two narrower surfaces held parallel with the ear. This is only a minor technical point and does not in any way affect the value of Dr. Macfarlan's interesting communication regarding an unusual case and its possible implications.

DR. J. GORDON WILSON, Chicago: Dr. Macfarlan has presented a study of a case of great deficiency of hearing, but not complete deafness, in which he was led to believe that there was present what one might call tactile hearing as distinguished from touch, a case in which the tactile sense may be supposed to have stimulated a cerebral center associated with audition. I suggest that in this case one is dealing with a remnant of hearing, regarding the inherent possibility of which there is much to learn. It is well, however, to have a case of this kind brought to the attention of otologists, for, as Dr. Macfarlan said, it furnishes an example of a center of auditory perception aroused by sensory stimuli.

DR. STACY R. GUILD, Baltimore: The publication of Strehl about 1895 and that of W. S. Bryant about 1910 have a bearing on the case which Dr. Macfarlan has presented. Both reported observations on hearing by means of tactile perception, and Bryant suggested the term pallesthesia for the phenomenon.

PROF. JOHN TAIT, Montreal, Canada: In our laboratory we have tried to distinguish between tremor sensation and auditory sense. My conception is that these things may be represented in different parts of the cerebral cortex. In Pavlov's laboratory it was found necessary to destroy a very large region of the cortex of dogs in order to render the animals completely deaf. Meantime, Munk had localized the auditory region in a much smaller area. We found that when the Munk area is ablated a dog no longer responds to sound, as tested by conditioning, yet retains a conditioned response to tremor.

It is just possible that the pacinian corpuscles, abundantly present in the fingertips, are the actual tremor receptors. They are present in the interosseous membranes, and, what is still more significant, they occur in the mesentery of all true cats. Cats purr and, for all one knows, may derive some special sensation from the mesentery during purring. Dr. Macfarlan's patient evidently had a remnant of hearing. As I understand him, Dr. Macfarlan would perhaps suggest that the patient first registers tremor sensation in a cortical tremor area. By association fibers this is then transmitted into an actual auditory sensation. The difficulty here, as Dr. Bunch suggests, is that this conception goes against the doctrine of specific nerve energy.

DR. J. GORDON WILSON, Chicago: In the drum of the ear there are no pacinian corpuscles. There are only pain endings.

DR. EDMUND P. FOWLER, New York: In a totally deaf ear one often notes the Weber phenomenon. I had one patient who was totally deaf in one ear. The whole labyrinth had sloughed out, but in the low tones he referred the Weber test to that ear. He evidently obtained some sort of sensation there. Perhaps the mucous membrane of the middle ear was more sensitive to the tactile impacts. Another difficulty is that of masking an ear when both are deafened. Masking sometimes produces a Weber phenomenon referred to the masked ear.

DR. DOUGLAS MACFARLAN, Philadelphia: I hope the members do not think that I was too hardy in suggesting an explanation. My idea is that one can do much with the residual hearing by training. Gault's work has been that of educating the tactile sense. He uses five telephone receivers grouped into one small instrument. He trains patients to pick up tactile sensations from the fingertips placed on the receivers. Each receiver passes only a certain frequency band. Compared with lip reading this method offers little for education in language. It does not give the patient as much appreciation of speech as he obtains from watching a man's face.

Musicians who are deaf use bone conduction. As Beethoven lost his hearing, he used to place his forehead on the piano. Later, he lost all hearing and the conception he obtained of his music was entirely subjective. In this synthesis the psychic and spiritual spheres alone took part. The severely deaf cannot get satisfactory impressions of tones by bone conduction but the psychologic factor of appreciation of rhythm which remains in the tactile sense is enormous. Helen Keller senses rhythm and vibration.

My paper attempts to show that in tactile sensation there remain what can be recognized as the beginnings of the special sense of hearing. The stimuli that I used were too weak to give bone conduction to the end-organ.

The question of training is important. Parrel, Marage and Cathcart have done convincing work on training remnants of hearing. I do not believe that this work will lead to hearing through the finger-tips. The laity have become interested and have invented all kinds of amplifiers, but the special senses are too exquisitely differentiated for one to presuppose that the crude tactile sense can be trained to take the place of hearing.

This is the only case in which I know there was a definite auditory image. It is interesting, but one could not let the patient give up lip reading for an ephemeral thing of this sort.

SOME EXPERIMENTS ON THE UTRICLE. DR. W. J. MCNALLY, Montreal, Canada.

In this short communication two main groups of experiments from a joint research with Prof. John Tait of the department of physiology of McGill University are considered. In the first group of experiments the utricles were intact and all other labyrinthine structures were removed. When only two utricles remained the frog showed marked swaying and pendulation. These oscillations sometimes occurred over a dozen times before the animal finally came to rest. When quickly tilted the body of the frog moved in the direction of the tilt and therefore in the opposite direction to that of true compensation. If the two-utricled frog was slowly tilted, it made a true compensatory movement but in definite interrupted shifts. The righting reflexes were still present. If one of the two remaining utricles was removed, the frog showed a lean toward the side of the absent urticle. However, if the head was carefully manipulated the lean could be undone and the animal returned to symmetry. The symmetrical pose was maintained until the supporting table was jarred or until the animal made a spontaneous movement.

In the second group of experiments the utricles were removed and all other labyrinthine structures were intact. The righting reflex was present and prompt in this de-utriculate frog. During movement the frog showed a decided tremor somewhat resembling that of paralysis agitans. Sustained forward progression was interfered with. The responses to quick tilting were marked. They were in the direction of true compensation but were greatly overdone. If only one utricle was removed and the frog was subjected to quick tilting toward the side operated on, the responses from the vertical canals were normal. If the frog was tilted to the other side the responses from the vertical canals were much overdone but were in the direction of true compensation.

The following conclusions are drawn: The utricles are not an essential part of the righting mechanism in the frog. They are necessary for sustained forward progression. The adequate stimulus of the utricular macula is probably not the pull or pressure of its otolith but rather some slight displacement of the otolith. The utricle is stimulated by any sudden tipping movement, and the reaction elicited may be described as negative or opposite to true compensation. The negative reaction and the existence of pendulation in the frog with only the utricles intact suggest that normally these reactions must be checked by the canals. The unduly strong reactions from the vertical canals of the de-utriculate frog to sudden tilting suggest a regulating influence of the utricles over the canals in the normal frog. Since the reactions of the utricle or of a canal to a quick tilt in a certain direction are oppositely directed, it is probable that in the normal animal the utricle and the canal oppose each other, the effect of the canal predominating, so that just the exact amount of compensation is rapidly effected.

DISCUSSION

PROF. JOHN TAIT, Montreal, Canada: Dr. McNally began practicing labyrinthine operations in 1924, and has practiced steadily ever since. As a result he can now with perfect certainty do either of two things to a frog. He can

either leave a pair of utricular maculae (or one single macula) behind, having put all other labyrinthine receptors out of commission, or do a thing that is still more difficult, viz., perform uncomplicated severance of the nerves to the utricular maculae without damage to any other receptors. It is due entirely to the unusual skill that he has acquired in these operations—operations never done before—that he is able to give the report that he has furnished.

DR. ELLISON L. ROSS, Chicago: The approach to the utricle is extremely difficult. Anatomically it is most secluded. It is closely guarded on all sides by parts which have great influences on equilibrium and the functions of which are not perfectly understood.

There are some advantages in the anatomy of the vestibular nerve. It is divided into two parts. One of these parts supplies the horizontal anterior semicircular canals, and the other, the posterior canals and the utricle. Therefore, following an injury to the first part of the nerve equilibrium would be profoundly disturbed when quick movements of the body were made. If the second part of the nerve was altered, movements in the planes of the vertical canals would produce a disturbance of equilibrium, but only to a minor degree. In addition, according to Dr. NcNally, the equilibrium of the subject in a state of rest would be destroyed.

Mistakes are often made in the diagnosis of cases of vertigo. There may be no history of a sense of rotation which is aggravated by changes of position. The rotation and caloric tests may give negative results. Are not such patients too often placed outside the class with vestibular disturbances? If the patient complains of a sensation of falling or of inability to sit upright steadily, may there not be a lesion in the utricle or in the nerve fibers leading from it?

I should like to ask Dr. McNally if there is nausea associated with a pathologic process in the utricle.

DR. W. J. MCNALLY, Montreal, Canada: In reply to Dr. Ross, it is highly desirable to arrive at some clinical application of these findings, but at present little is known about the subject, and the work is still in the experimental stage. The facts discovered in the frog must be shown to hold true for the higher vertebrates and finally for man.

Evening Session, May 8, 1933

A CLINICAL CONTRIBUTION TO THE STUDY OF MUSCULAR SPASM OCCURRING DURING RECOVERY FROM FACIAL PALSY. DR. ARTHUR B. DUEL, New York.

My pleasure in presenting this paper is not unmingled with anxiety. With Sir Charles Ballance I worked for a long time in the effort to introduce a method of direct repair of the facial nerve. The method achieved definite success, but in the course of recovery, certain phenomena arose which led me to address this body of otologists on a neurologic and physiologic subject. It may therefore appear somewhat audacious for me to present my theory for these phenomena, which is based purely on clinical facts.

Two years ago, Sir Charles and I presented the results of an investigation on baboons which proved that the direct line graft was superior to other methods of anastomosis. In the final experiment, following which all of the animals were killed, operation disclosed that the facial musculature had been restored.

In several instances, we allowed animals to recover from anesthesia and found that many of them exhibited facial spasm, tic or associated movements of the face. When the animals were finally killed we removed the cranium and stimulated the facial area down to the striate levels and to the facial nucleus. Then we dissected the tissue down to the base of the stylomastoid foramen to follow the tract of the restored facial nerve.

Sir Charles and I later showed thirteen human subjects in whom our method had been employed successfully. One difficulty, however, was that there was a long period of waiting from the time of the graft implant until nerve impulses

came through the face and until recovery, which was partial or nearly complete. None of the patients showed any facial spasm or associated movement in either groups of muscles after a year.

We next attempted to improve the technic by using a "prepared" graft, produced by degeneration in situ with a result similar to wallerian degeneration as it takes place in the nerve in cases of Bell's palsy. The animals used for this work were rhesus monkeys. The experiment necessitated frequent capture of the animals, placing them on the table and stimulation with the faradic current to see how early restoration of muscular function occurred. In a series of twenty-five monkeys it was found to take place at about the same time that conjunctival irritation (the winking reflex) reappeared. Careful observation shows that facial spasm occurred in the caged animals during excitement, fright and other emotional reactions. If, however, anesthesia was carried to a point where the conjunctival reflex was abolished, the facial spasm failed to appear, even on faradic stimulation.

Further work followed on the physiologic and neurologic phenomena of motor impulses. Let it be said here that there are reflex responses, such as the winking reflex, which are quite involuntary. The grin of the monkey and its expression of fear, anger or surprise are involuntary. In man, however, the winking reflex can be consciously controlled by the higher centers.

In regard to the mechanism of these impulses, in man and in the higher apes there is an area in front of the fissure of Rolando, in the precentral gyrus, in which muscular control of the body takes place. As that area is stimulated from above downward, one obtains movements first of the limbs, then of the face, then of the tongue, etc. The areas for muscular control are well mapped out in the cortex. The impulses to the face pass down through the genu of the internal capsule to the facial muscles in the pons. The other impulses penetrate to a point below the cortex where one can stimulate the striate level and obtain movements of the face. The impulses of the conjunctivae pass into the facial nucleus which receives the impulses which are to be transmitted. The cortical or controlling impulses come into this receiving box, and the striate level brings in all impressions of the senses but nothing volitional. The result is the balance of all impulses—striate and cortical—which are coming in. The system which controls the display of emotional expression is called archeokinetic. The lower or involuntary system is called paleokinetic. The facial nerve is the common pathway of these systems.

One must now ask: 1. Why did the animals, at a certain time, display these phenomena? 2. Why was the facial spasm obliterated under deep anesthesia? To answer these questions I uncovered the cortical voluntary areas in a monkey and, under anesthesia, stimulated a mapped-out facial area after it was well delineated. I found first the face and then the tongue areas. I then closed up the wound. The animal fully recovered, and there was no facial palsy. He responded to all types of emotional expression. If, however, the cortical centers which control the food pouches were injured the animal was unable to squeeze the food into the mouth on the injured side. He appeared surprised and then pushed the food from the pouch into the mouth with his fists.

The object of this test was to get the animal to recover without cortical control and then obtain facial repair as I had in others with cortical control. The animal thus recovered his emotional and reflex movements. When the conjunctival reflex returned, the animal had tic, as the others had. This proves that there can be muscular spasm or tic without any of the cortical control centers. I still have a few animals of this series, all of which, in the course of recovery, exhibited a muscle spasm at the same stage. This spasm is not related to cortical control.

In man, at a certain stage of recovery, there are associated movements. The reason for this is that the summation of all impulses passes through the facial nucleus to make a response. The facial nerve is made up of a system of neurons, the business of which is to convey these messages to the face. These neurons are of different types; certain neurons convey mass movements to the face, but certain higher types control isolated areas of the face. In normal nerves the

muscular response is smooth, even and synchronous on both sides. In the injured nerve all the injured nerve cells in the distal segment have to be carried away before repair can take place from the proximal end. The neurons push through the graft in a less orderly fashion than that of normal growth. The fast growing, nonmedullated types get ahead, and mass muscular movements occur because there are not enough of the higher myelinated fibers to assume control in isolated areas. The muscular twitches and associated movements are the result of lack of cortical control.

DISCUSSION

DR. J. RAMSAY HUNT, New York: The work of Dr. Duel constitutes a real advance in that it does not require switching, as in hypoglossal anastomosis. The facial spasms and twitches noted do not surprise me. Neurologists see them constantly in the moderate and severe cases; only the very mild cases are free from them. The first evidence of recovery is a toning up of the face. It does not sag so much. The first movement is the closing of the eye with movement of the angle of the mouth, which becomes more marked as more power is acquired. Then there is the phenomenon of overexcitability of the whole facial musculature. The whole side of the face contracts. Motility in the man is a very complex affair. The phylogenetic development of the nervous system is an increasingly complex process. The lowest forms show reflexes related to the segmental or archeokinetic nervous system. Later it becomes necessary to harmonize the reflexes, and the paleopyramidal or extrapyramidal system comes into play, the centers for the latter being at the thalamostriate level. The thalamic level represents the sensory system, and the striatal level, the motor system. The spinal cord represents a system governing associated movements, such as running, walking, advance, orientation and facial expression. The next development is the pyramidal tract or corticospinal system. Impulses of the rolandic area of the cortex pass down through the capsule, through the whole length of the brain stem and spinal cord to the anterior horn cells. This is the neokinetic system, and it exercises control over isolated movement. It has taken millions of years of development to produce such a system, which, like the others, follows an important law, that each system governs the one immediately below it. During regeneration, the lower type of system regenerates more rapidly, but it takes longer for the more highly specialized cells to be replaced and assume their powers of control.

DR. J. GORDON WILSON, Chicago: Dr. Duel advances as a result of his experimental observations the hypothesis that facial spasm may also be due to faulty conveyance of impulses. He produces evidence to show that the causation may be in the nerve itself. In support of his hypothesis there is the fact that facial spasm has been observed in traumatic irritation of the trunk of the seventh nerve, as in aural lesions or compression of the seventh nerve by a tumor, or in both preceding facial paralysis. The experimental evidence that Dr. Duel has presented seems convincing that it may come from faulty conveyance of impulses. As the nerve grows down from the center seeking the peripheral stump it comes in contact with the scar arising from the section and there forms a mesh of nerve fibers. Some of these form associations far apart from those which they had before section of the nerve. They wander into peripheral paths not associated with the muscle with which they previously were associated physiologically, and so the anatomically associated group of neurons has to learn a new physiologic association. Till this is done a number of nonassociated muscle groups are involved in the nerve impulse. This theory appears to me to offer a more adequate explanation than that of spread through inadequately insulated fibers.

That a nerve recovering from paralysis can exhibit a spread to parts not originally associated with the impulse gains support from the union of sectioned sensory nerves. Thus a recovering sensory nerve head shows that in a hypesthetic area in the skin there may be a spread of sensation to parts not definitely involved in the localized cutaneous irritation. This appears to me to be the same phenomenon looked at from the other end of the nerve. This local spread may be due to

imperfect conduction in the nerve, as Dr. Duel has suggested, or to lack of control in more specialized cutaneous structure, as Dr. Hunt would suggest.

Dr. I. D. Kelley, Jr., St. Louis: I have operated in one clinical case in which the patient is now on the road to recovery. I was impressed with the necessity for developing a definite operative technic before undertaking this operation for repair of the facial nerve in man. I should caution otologists before they undertake this operation either to copy Dr. Duel's technic, evolved through long experience in the laboratory and on the cadaver, or to develop for themselves through preliminary work on cadavers an individual operative technic of their own. Otologists, I believe, have been universally taught to stay away from the facial nerve as far as possible in their operations on the mastoid. Many have seen the facial nerve in the lining only when it has been pathologically or accidentally uncovered, and are quite unfamiliar with its course from the geniculate ganglion to the mastoid as it passes through the fallopian canal. The technic of dealing with the nerve at the site of injury and the introduction of the prepared autoplastic graft into position should be so perfected before operation as not only to insure the viability of the graft but to reduce the operative time, which at best is uncomfortably long for both the operator and the patient.

At present I am conducting a series of experiments on the timing mechanism of the laryngeal valves, using the Ballance-Duel method of repair of nerves, in the hope of ultimately correcting paralysis of the vocal cords. This experimental work is being done on dogs because of their loud bark and mobile larynx. In each dog one or both recurrent laryngeal nerves are severed below their entrance into the larynx. Certain of these nerves are immediately sutured and their cut ends joined together, while others are left with their proximal and distal ends free to be united only when the wound in the neck has healed and after degeneration of the distal nerve is complete. By means of a prepared autoplastic graft these nerves are then repaired in all cases, even in dogs in which primary union and regeneration of the nerve were permitted. At operation and during the period of degeneration and regeneration frequent laryngoscopic examinations are made to determine what changes take place in laryngeal function, whether repair by direct union or autoplastic graft accomplishes the best result and how near to normal the restoration of vocal cord function is when the repair of the nerves is finally complete.

Because of the necessity of perfect timing of the vocal cords in the production of voice, I think that the Ballance-Duel method of confusion of associated actions which resulted in nerve anastomoses, as first employed by Sir Charles Ballance, should be studied. Should muscular spasm or faulty timing occur here as in repair of the facial nerve during regeneration it then can be attributed directly to an interference with the nerve itself or its centers. As Dr. Duel has just shown, one should expect no permanent injury to the laryngeal valves resulting in malfunction but eventually the restoration of normal voice. Such findings would make operations on the recurrent laryngeal nerves as imperative as those on the facial nerve. I hope to present these results later.

Several significant facts were demonstrated to me in reviewing Dr. Duel's work: First, the facial spasms occurring in all of these cases appear at about the same time, when a response to the conjunctival irritation or threat appears; second, this muscular spasm increases as the improvement progresses, but only to a certain point, when it becomes less evident. When I saw these cases they had not been observed long enough to determine whether the spasm had entirely disappeared, although in a number of instances it had grown progressively less. In persons who have muscular spasm during the course of recovery, disappearance of the spasm should be considered a great moment in the progress of nerve regeneration.

In studies of the rhesus monkey which shows normal emotional reflexes following the removal of both cortical voluntary centers and in which an involuntary muscular spasm develops only after an implantation of an autoplastic graft in the facial nerve, and then only when restoration of function has begun, it would

appear that Dr. Duel is correct in assuming that the spasm in the muscle must be due to inadequate transmission of impulses along the nerve through some faulty mechanism of repair.

Morning Session, May 9, 1933

FURTHER ANALYSIS OF COCHLEAR ACTIVITY AND AUDITORY ACTION CURRENTS (WITH DEMONSTRATION OF AUDIBLE RESPONSE). DR. H. DAVIS, DR. A. J. DERBYSHIRE, DR. MOSES H. LURIE and DR. L. J. SAUL, Boston.

The electric response of the cochlea to stimulation by sound was led from the round window of a cat to a suitable amplifier and studied by means of a loud speaker and a cathode ray oscillograph. This response can probably be used with some confidence as an index of mechanical disturbances taking place in the inner ear, since it reproduces accurately sine waves of all frequencies in the audible range within physiologic limits of intensity. The intensity of the response is roughly proportional to the logarithm of the intensity of the stimulus, but deviates from this law at very low and very high intensities. It shows no fatigue except at very high intensities, when action currents of nerves may constitute part of the response. The threshold, as observed by the cathode ray oscillograph, corresponds qualitatively, and in favorable preparations almost quantitatively, with the human audibility curve. When a stimulating wave of low frequency containing higher harmonics is used, the harmonics are reproduced relatively more strongly than the fundamental tone. When two different tones are presented simultaneously, new frequencies corresponding to the sum and to the difference of the original frequencies appear in the response.

It has been impossible to study auditory action currents satisfactorily in the eighth nerve, owing to the inevitable admixture of cochlear response. They may be obtained in pure form if coaxial electrodes are placed in the brachium of the inferior colliculus. Here one finds: (1) synchronized action currents reproducing the frequency of the stimulating tone; (2) asynchronous action currents, persisting as an irregular random discharge throughout stimulation, and (3) an "on effect," a large outburst of activity associated with the sudden beginning of a tone or noise. All three types of response show fatigue. The upper limit of frequency reproduced in the brachium is 2,400 cycles per second, but asynchronous activity is also present above 1,000 cycles per second and increases in proportion as the frequency is raised. In favorable preparations the threshold for detection of action currents may be nearly as low as that for the cochlear response, and the relations of this threshold to frequency, and of magnitude of response to intensity, resemble in broad outline the relationships of the cochlear response. This tends to justify the use of the cochlear response as an indicator of the activity of the auditory mechanism in the study of normal animals.

DISCUSSION

DR. SAMUEL J. CROWE, Baltimore: Dr. Davis and his associates have shown that auditory action currents may be detected and studied only in the central auditory pathways. An electrode placed on the auditory nerve at any point between the brain stem and the temporal bone will pick up both the action current and a peculiar electric response of unknown origin, which is called the "cochlear response" or "the spread." I shall let the physiologists discuss the auditory action currents picked up near the inferior colliculus, which have a frequency of 2,400 cycles. I have had some experience, however, with the other type of response of an animal's ear to sound stimuli, which is called "cochlear activity" or "spread." I shall briefly review some of my reasons for thinking that it is a reliable index of the functional condition of the middle and inner ear and a reliable method for the experimental study of the physiology of the ear.

1. With an electrode on both the right and the left auditory nerves the transmission of sound is equally good on the two sides, provided both ears are normal.

2. If there is otitis media on one side the transmission of speech is much diminished and the lower frequency tones up to 1,500 are much impaired on that side but normal on the unaffected side.

3. If there is a labyrinthitis on one side no sound is transmitted on the affected side while there is a normal response on the side of the normal middle and inner ear.

4. In a series of albino cats no sound of any kind was transmitted by either ear, and histologic sections of the cochlea showed everything normal but the organ of Corti and the stria vascularis. In these albino cats the utriculus and cristae of the canals are normal, but the sacculus is atrophic. In other words, a normal organ of Corti is necessary for the transmission of sound of any kind through the cat's ear by the Wever and Bray method.

5. Division of the ossicles immediately impairs greatly the transmission of the spoken voice and particularly the low tones.

6. Fixation of the ossicular chain by means of tension on the tendon of the tensor tympani reduces the transmission of the voice and pure tones, but this promptly returns to normal when the tension is released (provided no damage has been done to the ossicles or their joints by excessive tension).

7. If a graft is placed in the round window niche on one side only, and two months later the animal is tested with an electrode placed on both the right and the left auditory nerves, it is always found that the ear that was operated on transmits the voice and all tones from 50 to 100 per cent better (louder, clearer, more resonant) than the normal ear.

8. If the bulla is opened and a preliminary test is made for the transmission of voice and all tones from 250 to 8,000 double vibrations, a small opening is made in the membrane of the round window with a needle and the tests for voice and tones are repeated several times, it is invariably true that as the perilymph escapes, the transmission becomes more and more impaired.

9. It has been shown many times that a marked increase in the pressure of the perilymph has no effect on the transmission of sound, but a decrease always affects the transmission of sound, and the high tones are the first to be lost.

10. Localized cochlear lesions, made by a method that prevents the escape of endolymph or perilymph and avoids intracochlear bleeding, are followed by a complete loss of transmission of some frequencies while all the others are preserved.

11. Following the experiments the results were checked by means of serial sections of the middle and inner ear.

For these reasons it is considered that whatever the nature of the cochlear response is it is a reliable index of the functional activity of the middle ear. The experiments of Dr. Davis and his associates have been largely confined to the study of the central auditory pathways while the foregoing studies were limited to that part of the auditory apparatus that lies within the temporal bone.

DR. E. A. CULLER, Champaign, Ill.: There are one or two points I should like to bring out. If, as Dr. Davis maintains, frequencies above 2,500 cycles cannot be transmitted through the auditory tract, how does the animal distinguish these higher frequencies (say, 5,000 cycles) from low frequencies of the pitch that was actually heard coming out of the loud speaker?

In my studies of auditory acuity there is emphasized the "conditioned response" method, by which it is possible to measure an animal's hearing directly. The animal is first trained to respond every time he hears the stimulus. This is done by first sounding the tone for two seconds and then administering a shock to the right forepaw just strong enough to effect quick withdrawal of the foot. After a few days, the animal begins to withdraw his feet as soon as the tone begins and thus avoids the approaching shock. The method requires patience, but it works well. After five or six weeks of training the dog is ready for accurate testing. If the tone is made fainter and fainter, he continues to react as long as he hears it; when it becomes inaudible, he no longer responds. Auditory acuity in a dog thus trained may be measured with as great precision and consistency as can that of a human being. I say this because an animal's mean variation over

a period of twenty or thirty days will not exceed 1 or 2 decibels. I do not believe that the average human subject would show more uniformity than that.

I consider these measures of actual hearing particularly important in connection with current problems. For example, is the electric response which can be picked up from the cochlea and the eighth nerve truly auditory? This question, in my opinion, is reduced to another: Does the electric response correlate with the animal's hearing? If so, then the electric response is a truly auditory phenomenon; otherwise, it is not. In a like manner the striking results of Dr. Hughson and Dr. Crowe led me to inquire: If the round window is blocked, will the animal actually hear better? On using a plug of soft gum, I found both that the animal's hearing was impaired and that the electric pick-up from the eighth nerve was reduced. I ascribe this discrepancy in the results largely to the type of plug used. Finally, in connection with the effect of x-rays on hearing, there is evidence that when mild doses (less than 100 roentgens) are applied to the whole auditory tract of a dog, acuity of hearing will increase about 5 decibels above the normal level. Here again it is hoped to correlate this gain with changes in the electric response from the cortex and midbrain.

In conclusion, I believe that the ultimate criterion in deciding whether a given phenomenon is truly auditory must be sought in actual tests of hearing in the animal, however fascinating and important the electric response and other changes may be.

DR. WALTER HUGHSON, Baltimore: I should like to ask Dr. Davis if he is willing to commit himself as to the nature of the cochlear effect. This phenomenon should be called the cochlear effect rather than the pick-up, as the term pick-up is used constantly to describe magnetic interference of a purely electric nature. I should like to know whether they have found any difference in the intensity of response related directly to the distance of the electrode from the internal auditory meatus. Is the response due to this distance or can it be explained on an anatomic basis? In my experience the cochlear effect is influenced by exactly the same things that influence transmission through the nerve. With an electrode on the round window one can change the intensity of the tone transmitted by a variety of procedures. When a trephine opening is made in the cochlear wall, fluid escapes. Even with the escape of fluid the cochlear effect remains practically the same as it was before, while the response from the nerve diminishes rapidly.

DR. R. L. WEGEL, New York: I should like to speak of only one point, that of the upper limit of frequency transmissible as an action wave over the nerve trunk. A single nerve fiber is capable of transmitting a frequency not higher than a certain definite value. Dr. Davis observed that the nerve trunk transmits frequencies which are higher than one would suppose them to be. There is a reasonable tendency to suppose that the eighth nerve is so specialized as to work faster than other nerves of the same type; while this may be true, it is not a necessary deduction from the experiment. Indeed, the mechanism of mechanical stimulation points in another direction. It has been mentioned elsewhere that at the higher frequencies of vibration of the basilar membrane the stimulus sweeps along the membrane once during each cycle. This should tend to excite accessible elements of nerve tissue progressively, beginning at the proximal end and proceeding with increasing amplitude to a certain region and then decreasing to the distal end. Thus, regardless of the number of functional contacts made by each fibril on the hair cells (provided only that each fibril does not contact every hair cell), there must result a progressive excitation of tissue for each cycle and a tendency to echelon formation of fiber action wave fronts proceeding along the trunk. The highest measurable frequency on this trunk is then equal to the highest frequency possible for one fiber multiplied by the greatest number of instrumentally detectable steps of the single echelon. Dr. Davis was unable to detect a higher frequency than 2,500 cycles, and the limiting fiber frequency is said to be about half this figure, so it would seem that only two steps per echelon rank are at present measurable. The highest observable frequency on a nerve trunk must depend in a measure on certain now unknown properties of the mechanism of detection by

apparatus. A definite figure given for the frequency limit of a nerve trunk is too easily misinterpreted to be of purely physiologic significance. I am therefore inclined to suggest that this question be viewed like that of the fictitious "upper" and "lower" frequency limits of hearing and that the attempt be made, if it is considered practicable, to determine the rate at which this effect decreases with a frequency near the supposed upper limit. The existence of such a rate as a specific property of the mechanism will probably not be questioned. Its value must then enter somewhere into a comprehensive understanding of the "theory" of hearing.

Dr. H. Davis, Boston: I am glad that Dr. Crowe has discussed the results obtained in his laboratory. When his results are compared with ours the details seem to unite in giving a better picture. I agree that the electric effect from the cochlea reveals mechanical events within the ear. It looks more and more as if it depended on the activity of the organ of Corti. Dr. Crowe spoke of failing to obtain a response in the ear of the congenitally deaf albino cat. These animals lack the organ of Corti. That is most significant. We planned the same experiment, but lost both time and animals trying to breed them. Another point is that localization of lesions in the basilar membrane is a fundamental point and must be carried out with the greatest care. One will then have a direct demonstration that perception of a given pitch depends on the integrity of a certain portion of the basilar membrane. Such an association for the high frequencies has been established by Dr. Guild's work. To extend that to the whole auditory range would give a clear picture of the mechanism of the inner ear. I am inclined to agree with Dr. Williams as to the nature of the cochlear response, that is, that the actual sensory cells of the organ of Corti are responsible. I do not know exactly how it works, but it seems to be the essential anatomic structure. This is in agreement with many other observations. With regard to Dr. Culler's question, one must study responses as well as ears in order to understand auditory phenomena. We hope ultimately to understand the type of disturbance that underlies sensation and to correlate it with our subjective interpretations. We can have confidence in a conditioned reflex, used as Dr. Culler has used it, as an index of auditory acuity.

Fixation of the round window is a difficult point to interpret. We have had difficulty in getting a clearcut experiment, and are not yet satisfied with our observations. They need particularly careful interpretation. In regard to Dr. Hughson's question as to the distance from the cochlea, in a crude way the strength of the signal depends on the distance from the cochlea, but more accurately on the conditions of electric conductivity. When the electrodes are placed over tracts of some function other than auditory, one can hear a very fine spread, but plunging the electrode below the surface is sufficient to abolish this. The distance remains the same, but the interposition of membrane and fluids prevents the effect. I think that suffices to illustrate the point. In reply to Dr. Wegel, his concept of the physical activities of the basilar membrane has been the cornerstone of our research. We have tried to fulfil the theoretical predictions that he has made. We are not yet able to satisfy him on all points. We have not been able to find action currents spread out in time through a full half cycle of the stimulating wave, but we cannot say that such dispersion is absent.

Phylogenetic Development of the Vertebrate Cochlea. Dr. Moses H. Lurie, Boston.

The development of the cochlea is traced from the lowest to the highest of the vertebrates. Attention is called to the fact that the mammalian cochlea is the end-result of a double development for perception of sound. As the vertebrate animal leaves its aquatic environment to live in air the pars basilaris gradually develops in close relationship to the lagena. With higher development of the vertebrate the pars basilaris grows into the lagena, replaces it and takes over its function. In the lower forms of vertebrates only the lagena is present. It

has been shown that the lagena detects sounds of low frequency. In the higher forms in which the pars basillaris is present not only low but also high frequency sounds are heard.

Judging from its phylogenetic development the mammalian cochlea may function in two ways: 1. For sounds of low frequency the cochlea can respond as a whole. 2. For sounds of higher frequency the cochlea can respond at specific resonant points in the basilar membrane.

DISCUSSION

PROF. JOHN TAIT, Montreal, Canada: Dr. Lurie fixes his attention on two labyrinthine receptors which he seeks to trace in an upward direction. The lagena of fishes (otolithic) has been shown to pick up water-transmitted sounds. One must remember, too, that some of the sound receptors are exteroceptors, while others may be proprioceptors stimulated during emission of sound. In terrestrial vertebrates the pars basilaris appears. In the frog the pars basilaris rests on fixed structures. In higher vertebrates it is suspended, as it were, in the form of a real "vibrating" membrane. In reptiles and birds, the lagena and the pars basilaris tend to be approximated and to rest side by side. This is also seen, as Dr. Lurie has explained, in Monotremes. But in Eutheria or higher mammals the lagena has disappeared. His argument is that the eventual cochlear organ that originates from the fusion responds in two ways.

DR. LOUIS K. GUGGENHEIM, St. Louis: In the ontogenesis of the human labyrinth these various stages are passed through. When the ectoderm closes off as the auditory vesicle there is a structure lower than anything that Dr. Lurie has shown. At 31 days of age a dorsomesial outpouching (recessus labyrinthi-ductus endolymphaticus) occurs, a structure that is present in the lowest forms of life. At 45 days the vesicle shows constriction into the utriculus and sacculus. The evagination from the sacculus which will become the cochlea is similar to the lagena of fishes The wall of the evagination shows a thickening which will form the organ of Corti. Phylogenetically, the oval window appears first in the herring. I do not understand why fishes have an oval window. This structure is regarded as part of a mechanism designed to prepare certain sound waves for cochlear reception. The fish has no cochlea but simply a lagena for perception of water vibration. The beginning of the mammalian cochlea is the pars basilaris, which first appears in Amphibia.

DR. MOSES H. LURIE, Boston: All deaf people hear something in the cochlea. This residual hearing is the remnant of the primitive hearing sense. I do not know about the oval window. I have not worked on that aspect of the subject.

THE NEED FOR A REVISED NOMENCLATURE FOR CHRONIC PROGRESSIVE DEAFNESS. DR. THOMAS J. HARRIS, New York.

Dr. Harris calls attention to the fact that the present nomenclature is in need of revision to be in step with present-day views. That in use today dates back to the last thirty years of the last century, and is found in all the standard textbooks of that period, including those of Bezold, Gruber, Roosa and Politzer. Among these writers Politzer sets forth the grounds for such a classification most clearly. He expressed the belief that while it is possible for one form to be transformed into another, certain definite characteristics are present in types of disease of the middle ear from beginning to end. Politzer therefore maintains that the clinical basis is the only one for classification. In common with all other otologists of the period, he divided nonsuppurative diseases of the middle ear into two distinct groups. To the first he gave the name of "catarrh" of the middle ear, a condition which he recognized as involving both the middle ear and the eustachian tube and which is distinguished by a swelling of the mucous membrane with a throwing out of a serous or mucous secretion. Closely related to this first group of cases, but in many instances without any preceding secretion, is the second large

group which he called "dry catarrh" of the middle ear, an insidious process resulting in the formation of adhesions and the binding down of one or more of the ossicles. In Politzer's opinion, otosclerosis is a primary disease of the labyrinthine capsule, and ordinarily the middle ear is not involved.

Phillips, in the sixth edition of his book on "Diseases of the Ear, Nose and Throat" published in 1922, divided diseases of the middle ear into those which are nonbacterial and those which are bacterial in origin. The nonbacterial diseases of the middle ear, he stated, are known as "catarrhal" and those of bacterial origin as "inflammatory."

The catarrhal type is usually due to mechanical effects produced by closure of the eustachian tube, while those of bacterial origin are caused by the invading micro-organisms. Logan Turner and Fraser (1924) classified cases of deafness as "those caused by past attacks of catarrh or suppuration in the cleft of the middle ear, i. e., chronic and repeated processes in the middle ear or a cicatrized tympanum, and those due to otosclerosis." These quotations from present-day writers seem to convey the idea that the nomenclature so long in use is entirely satisfactory. The position, however, taken by Dwyer and by those who took part in the discussion of his paper four years ago is strongly opposed to this. In the discussion which followed, Duel also urged that the term "catarrhal" be dropped. He favored the term "hyperplastic" acute or chronic or atrophic acute or chronic, and emphasized the importance of the diagnosis of toxic infection of the eighth nerve. Emerson was of the opinion that a catarrhal process is a mild inflammation as compared with a more virulent attack involving the middle ear. In all cases the cause is a toxemia proceeding most often from the tonsils. In other words, in every infection that involves the middle ear, whether it is a catarrhal process or a more virulent attack, there is at the same time intermittent damage to the perception apparatus, in which the loss of bone conduction takes place in an orderly fashion.

The new nomenclature is based on etiology plus anatomic location. If a change is made in the present nomenclature, the new one must be one for use not merely in the office and the hospital, but in the clinic where the large number of patients seen does not permit of an extensive investigation to determine the diagnosis on entrance.

The classification adopted by the National Conference on Nomenclature may be the most satisfactory one. This can be determined only by suitable investigations. Dr. Harris would accordingly renew the recommendation made at Atlantic City four years ago, that a special committee be appointed to consider the proper nomenclature of diseases of the middle ear, and report at a future meeting, to act, if it seemed desirable, in conjunction with a similar committee of other national societies. The committee would also have as its task to say whether the form of progressive deafness commonly called otosclerosis is an actual entity, and, if so, whether involvement of the middle ear forms part of this entity, at least in a certain proportion of cases, or whether it is only coincidental with a process in the middle ear. Finally, it would be the duty of the committee to designate by a proper term that form of chronic deafness with recognizable changes in the middle ear which is at present called otitis media catarrhalis chronica, considered by every one to be a lazy unscientific term and one that calls for correction.

DISCUSSION

Dr. Arthur B. Duel, New York: Dr. Guggenheim made the statement that a tremendous number of cases of otosclerosis are found at autopsy which are never so diagnosed during life. It occurred to me that "otosclerosis" may be no more of an otologic term than are the words "gout" or "cerumen." When cerumen plugs the canal, there is deafness; also when otosclerosis attacks a certain part of the auditory mechanism, there is deafness. It would take some one wiser than I am to suggest a suitable nomenclature, but when others have built up a suitable system, I shall be glad to criticize it.

DR. S. J. KOPETZKY, New York: It is not necessary that a designation describe a disease. As long as we understand what we are talking about, that is sufficient. Specialists use many terms that are not listed in any system of nomenclature, and also encounter conditions for which there are no recognized terms. I am in accord with the recommendation of Dr. Harris that the Council appoint a committee to supply terms which otologists will agree to apply to the lesions with which they deal.

AN ANALYSIS OF A GROUP OF CHILDREN WITH IMPAIRED HEARING. DR. ALLAN WINTER ROWE, Boston.

A preliminary survey using the 4 *A* audiometer in a group of over two thousand children of school age showed nearly six hundred of them to be apparently hard of hearing. Two hundred and seventy-six children, however, had genuine impairment of hearing, as shown by individual study, including an examination of the ears, nose and throat, and audiograms obtained with the 2 *A* instrument. Careful psychometric examination of all but a few in the group demonstrated about fifty children with lowered intelligence. Thirty-six others required further study for reasons other than mental retardation. The present report deals with a diagnostic survey of this group of children, all of whom were hard of hearing and over half of whom had, in addition, mental retardation of varying degree. The two outstanding etiologic factors elicited by the study were lesions of the central nervous system of infective or traumatic origin and endocrinopathy. The evidence is discussed in detail, but as the data are chiefly significant only as they apply to the individual case, they are poorly adapted to abstract presentation. Bilateral loss of hearing was shown by 57 per cent of the group; in the remainder, only one ear was significantly affected.

DISCUSSION

DR. MARVIN JONES, New York: Dr. Hemsath has reported a high percentage of fatalities resulting from birth injury in proportion to the total death rate for infants. He procured and examined the temporal bones. Blood or exudate was found in the cochlea in some cases, and these findings will be reported in detail at some future time.

Crowe and Guild made an original observation in adult patients. The temporal bones of patients who had undergone craniotomy showed blood or exudate in the cochlea and its aqueduct.

One specimen obtained from a patient with a history of injury to the head showed exudate in the cochlea and an atrophy of Corti's organ. Considering these observations, it seems significant that deafness has been noted clinically following trauma of the head. Patients without demonstrable fracture of the skull may show a resultant loss of hearing some time after the accident.

Recent research has been directed toward endocrine dysfunction as a possible cause of deafness. Bailey considers the manifestation of thyroid dysfunction as the effect of toxemia. The toxemia produces a low normal reaction affecting incidentally the special senses. Patients having a low normal basal metabolic rate have improved generally on small doses of thyroid extract. The hearing probably improves, owing to the increased alertness of the patient. Children show a more pronounced reaction than do adults.

Mental retardation with associated deafness may be classified as of three types: (1) apparent mental deficiency due to traumatic or pathologic deafness, (2) mental deficiency due to deafness associated with disordered metabolism and (3) mental deficiency and deafness produced by developmental or degenerative changes in the central nervous system.

Otologists differentiate between these types of cases in order to advise treatment intelligently.

Obviously, the first two types are in the domain of the otologist. The mental mechanism is sound, but mental development has been retarded because of sensory defects. Education is the important phase of treatment, not the education of the child but that of the doctor, the parents and, last, but by no means least, the teacher.

To elucidate this point, the common reaction of the human mind, when faced with a problem, is a retreat rather than an attack. These children are a problem. When parents, teachers and doctors are faced with these difficult problems, their defense mechanism reacts to force the process of adjustment on the child. The child in turn reacts in its own defense and exhibits an unwillingness to accept the burden added to his sensory handicaps. He adopts a defense within himself by withdrawing from distasteful situations. An attempt to crash through this barrier results in an antagonism on his part, which is interpreted by the adults as noncooperation. Some of these defense devices are assumed stupidity, exaggerated deafness, aggressive attitudes, misguided humor, indifference or a general cantankerous deportment. Adults dread the study of children in their care, as they are often faced with a reflection of their own faults. These children are not bad; they are avoiding unpleasant contacts. If contacts are forced, they exhibit the reaction of a normal adult, for example, when he is ejected from the retreat of his afternoon nap.

Adults demand standard performances of children. Children who have sensory handicaps try to compensate for their impediments in order to produce normal responses. Sometimes they have sufficient mental capacity to compensate perfectly and produce a normal reaction. A normal response is possible only in a patient of higher than average mentality. More often the child fails in complete compensation, and is classed as mentally subnormal. The deaf child endeavors to compensate by lip reading and a development of hyperacuity of his normal senses, such as vision and touch.

By what approach may one hasten development in handicapped patients? The willingness to spend the time is essential. One must gain their confidence by approaching them through their own normal channels. One sees, for example, a child who is considered deaf and mentally retarded. Speech is lacking because he has never been able to hear. Communication is difficult. Subjects which depend on hearing and speech for communication are therefore not comprehended. In testing this child, one should use only methods which may be transmitted by the use of his normal senses. Frequently, these patients will show a hyperactive mentality. Mental perception may thus be proved normal and only the transmission at fault.

MR. H. M. MCMANAWAY, Staunton, Va.: I came to listen rather than to take part in the discussion. As an educator of the deaf, I hoped to learn much of value from this meeting. I cannot add anything from the medical standpoint, but there is one thing of practical value to you which I can say, that is in reference to intelligence tests in children with loss of hearing. Dr. Jones referred to difficulties which arose from tests of hearing through defective speech. One important thing which he did not mention and which I have been impressed with is the difficulty of performing intelligence tests on children with profound loss of hearing. In children seriously deaf from infancy or early childhood there is not only a lack of the knowledge of speech, but there is also a defect in the language ability of the child. The language defect is even more serious than the speech defect. The child does not understand either written or spoken language. Such children are often considered mentally defective because they cannot pass intelligence tests which for them are language tests, rather than tests of intelligence. This is not usually realized by the layman. May I then use a word of caution here and ask you to be careful not to class the child as mentally defective unless the test is other than a language test? In testing children who have been deaf since infancy, a form board or other performance test should be used, rather than a test involving the use of language. In administering the test, one should

be sure the child understands what is expected of him. Again, the young child's language deficiency may prevent him from understanding what he is expected to do; obviously failure would be due to his language handicap rather than to mental incapacity.

Dr. Edmund P. Fowler, New York: Eight years ago, we started our work with the audiometer. Dr. Fletcher's staff helped me with the tests, and I made otologic examinations of several thousand school children. In New York we have as yet no appropriation for this work, which is done by the New York League under great difficulties. Over a million children should be tested, but from a monetary standpoint there is no support in sight. I feel that Mr. McManaway's point is important. In many tests the child cannot respond because it has not heard sufficient language to understand the words. There is always a group of children who ought to be examined further. They are not necessarily mentally deficient. A large number of children are merely deficient in attention. That is a psychic factor. In regard to medicolegal cases, otologists often have to testify as to loss of hearing after concussion or bleeding from the ear.

Dr. Horace Newhart, Minneapolis: We have endeavored to make the public "ear-minded," but so far we have not succeeded in our ideal of reaching the whole population, although we have endeavored to test the children in all the schools. We lacked funds for propaganda and for cooperative work. We also lacked full cooperation from members of the medical profession. Probably the incidence of deafness in rural communities is much greater than one realizes. There is a lack of organized health service. The general practitioner does not realize the importance of hearing tests, and it is up to otologists to assume leadership in this matter.

Dr. Emil Amberg, Detroit: In my opinion there are five main points: educational, economic, medical, eugenic and social. In regard to the economic aspect of the question, I think that every state and community should give more consideration to the high cost of repeaters. One point that I should like to quote concerns the stigmas of degeneration. They are not very important except in connection with the whole picture. I have seen a high palate in many children in the Detroit Day School for the Deaf. My investigations in this respect have only comparative priority, as Hippocrates some twenty-three hundred years ago mentioned the combination of a high palate and disease of the ear. The social aspect is of more importance when the patient has grown up. In some instances to inquire whether the so-called mental inferiority or the impairment of hearing appeared first, is like asking which came first, the chicken or the egg.

I believe that head injuries form an etiologic factor in deafness. This is an important circumstance in Detroit because of the many automobiles there. In regard to deafness in rural schools, it is necessary that teachers of the deaf be given increased means to travel in order to do field work. Many children and adults who are now burdens to their families and to themselves could have a more normal and happy life if they were helped to overcome their handicaps and make social contacts.

Dr. Allan Winter Rowe, Boston: The basal metabolic rate in itself is an entirely insufficient ground on which to form a diagnostic estimate. In fact, broadly speaking, there is no one test which is competent to serve as the basis of a diagnosis. The figures reported for the metabolism rate are rarely truly basal. There are varying levels in the various endocrine disturbances, and extraneous elements all operate to raise the level rate and thus to yield potentially misleading results. Further, the normal standards used leave much to be desired, particularly in the case of younger persons and those whose physical configuration departs significantly from the normal. The Harris-Benedict standards are helpful, and my practice is to use both them and the Boothby modification of the Aub-DuBois standards and take the average of the two. In studying the patient one must obtain all of the measurements that one can; then each test, to assume its

full significance, must be interpreted in the light of all of the other evidence available.

Another factor of uncertainty is that one cannot rely on the report of the parent or guardian as to the behavior of the child. The parent may be unduly pessimistic or optimistic, and report the child's behavior in a form which is frankly misleading. Dr. Newhart raised the question of the relation of mental retardation to loss of hearing. My data are not competent to resolve this question, and it is difficult to analyze the available material. One should work with a selected group and carry out a series of correlated studies.

In regard to the question of eugenics, the status of the putative otosclerotic child is far from definitely established. I recently reviewed an elaborate eugenic investigation which carried the study of heredity over seven generations in individual families in the group, and yet the diagnosis of otosclerosis was not established in one of the children who formed the basis of this investigation. Naturally, if one is to study the hereditary influence in otosclerosis, the starting point is the child, and a proper otologic diagnosis must be established if the eugenic study is to have any meaning. In the elaborate investigation to which I refer this was not done, and there remains only a massive artefact of negligible significance.

Afternoon Session, May 9, 1933

THE MASTOID CELLS: THEIR ARRANGEMENT IN RELATION TO THE SIGMOID PORTION OF THE TRANSVERSE SINUS. DR. PHILIP E. MELTZER, Boston.

The contention is that mastoid cells, when present, are arranged in a systematic manner and this arrangement is dependent on the course and position that the lateral sinus assumes in relation to the cortex of the squama posterosuperiorly, the cortex of the mastoid laterally and posteriorly and the digastric crest or medial margin of the mastoid tip inferiorly, and in the relation to the posterior canal wall from the level of the lateral semicircular canal to the point where the canal wall merges with the medial or digastric crest of the tip. It does not matter whether the cells are large or small, pneumatic, diploic or mixed; the arrangement is systematic according to the relationship to the structures just mentioned.

The description of the cells is intended only to clarify the subject of this paper and not to add confusion to an already overtaxed nomenclature. One cannot improve on the classification of Mouret or Neumann.

The author distinguishes three groups of cells with their subdivisions: 1. The superior group and deep zygomatic cells: (*a*) the anterosuperior group, including the superficial cells, and (*b*) the posterosuperior group (angle, petrosal cells). These include the cells underlying the tegmen of the mastoideum and extending posteriorly from the antrum to the point where the lateral sinus joins the cortex of the squama.

2. The antero-inferior group: (*a*) the cells of the posterior canal wall and tip. These extend from the inferior margin of the antrum along the posterior canal wall into the tip and extend posteriorly to the point where the diagastric crest joins the cortex of the mastoid. These cells lie above a plane passing horizontal to the facial canal. (*b*) Retrofacial cells. These lie posterior and medial to the facial canal. Superiorly they come in contact with the deep subantral cells around the lateral and posterior semicircular canals, and inferiorly with the cells overlying the lateral sinus in the direction of the jugular bulb.

3. Mesioposterior group. These cells are the intermediate group lying interposed and coalescing with the superior and antero-inferior groups. They make up the greater mass of cells and lie in front, immediately over and posterior to the lateral sinus. The posterior cells are commonly designated the peripheral or marginal cells.

The other cells of the mastoid are not mentioned because they do not play a rôle in relation to the lateral sinus.

Observations were made of the relationship of the lateral sinus to these cells in many hundreds of temporal bones secured by dissection and by operation. These observations hold true in children as well as in adults.

As the lateral sinus is most variable in its position and in its relation to the cortex of the mastoid, posterior canal wall, digastric crest and other structures, a limited number of descriptions are given, sufficient only to show the trend of cellular arrangement.

The conclusions are: 1. It is evident that the lateral sinus does not influence the type of pneumatization; this is dependent on the character of the epithelium and subepithelial tissues.

2. It would appear that the position of the lateral sinus with reference to the structures mentioned determines to what extent the pneumatization will occur and causes a definite arrangement of the cells in relation to it.

3. Clinically, unless stereoscopic roentgenograms are made of the mastoid, the arrangement of cells as shown on the film will be indefinite, particularly the deep cells. Once the sinus plate is exposed and its position in relation to the structures named is noted the cellular pattern of the mastoid becomes known, and it will be nothing short of negligence to fail to uncover the remotest cell.

4. The course and relations of the lateral sinus offer to the surgeon an interior landmark of the mastoid structures comparable in importance to the external landmarks used as a guide in locating the antrum.

DISCUSSION

Dr. James A. Babbitt, Philadelphia: Dr. Meltzer has given a clearcut description of pneumatization and alteration of the cells under inflammatory influence and of the arrangement of the cells. He then proceeds under a new and somewhat baffling nomenclature to make a classification of the cells in their relation to the lateral sinus and particularly the sinus plate. According to my interpretation of his paper, a basic point is the contention that the contact relations of this plate to the squama, digastric groove, canal wall and posterior cortex forestall further pneumatic development. Finally, in his conclusions he maintains that the development of these pneumatic cells is conditioned by the position of the lateral sinus, and knowledge of the regularity in this arrangement should infallibly guide one in complete operations.

The real kernel in this paper is the correlation in the arrangement of pneumatized cells. Stereoscopic examination, which is the rule in well ordered departments of roentgenology, should reveal this in any event. Regardless of this, careful exposure of the plate and adequate study would indicate the position of all existent cells and should prevent failure in their eradication. Does, however, the discovery of this correlation in arrangement warrant the conclusion that the lateral sinus determines the extent of pneumatization and definite arrangement in relation to it? Furthermore, the clinical value of ability to uncover the remotest cell is stressed in this knowledge of arrangement. May one have the temerity to suggest that history has convinced every experienced operator that operations on the mastoid can sometimes be carried too far and that after a perfect clinical result roentgenograms will often reveal unexplored cells?

Dr. H. P. Mosher, Boston: This paper is a valuable and systematic presentation of the arrangement of the mastoid cells. A working knowledge of the position of the cells leads to a definite plan of operation and makes the mastoid operation a craftsman-like affair.

I am sorry that Dr. Meltzer thought that he had to change the nomenclature. Simplification is a greater help than elaboration.

The position of the lateral sinus is associated with the presence or absence of certain groups of the cells. Formerly the operator could make no guess as to the grouping until the cortex of the mastoid was removed. Today the roent-

genogram gives this preoperative information. To me this is one of the greatest things which x-rays do in connection with the mastoid, whether it is healthy or diseased.

It would seem that the position of the sinus determined the position of the cells, but it is possible to argue the other way around and say that the way the cells develop determines the position of the sinus. As the sinus is more important in the function of the body than the mastoid cells, perhaps it is more logical to give the determining rôle to the sinus.

Dr. Robert J. Hunter, Philadelphia: The points brought up by Dr. Meltzer are of great practical importance. A great deal has been said at this session about phylogenetic development. The temporal bone results from a fusion of the squamous and petrous bones and the tympanic ring. The mastoid process of the temporal bone is formed in part by cells from the squamosa and in part by cells from the petrosa. If one will imagine lava flowing over petrous rock one will have a good conception of the development of the mastoid process. The petrosquamosal suture marks the place where the squamous part of the mastoid process joins the petrous part. Beginning at this point and parallel to the axis of the petrous portion of the temporal bone, Körner has described a septum which divides the process into two parts. These parts vary in relative size in different persons. They pneumatize at a different rate. In one person the petrous portion is overdeveloped and in another the squamous portion. The squamous portion may be diploic while the petrous is cellular; this accounts for the fact that one may penetrate normal bone in a mastoid operation and find pus around the sinus. The infection may go down through the squamosal antrum or the petrosal antrum mentioned by Dr. Babbitt and therefore affect only one of the aforementioned systems of cells. The phenomenon noted by Dr. Almour, as described by Dr. Kopetzky, is explained in this way.

Dr. S. J. Kopetzky, New York: I appreciate this work on the presence, classification and arrangement of cells in the mastoid process. The work on arrangement will be justified in time. Dr. Babbitt referred to the presentation before the New York Academy of Medicine recently in which Dr. Almour, of New York, presented a study (which I here want to record because of its priority) of the development of a series of cells along the inferior and retrofacial groups which form the cells of the tip. Dr. Almour made a simple experiment. He shut off the mastoid antrum from this accessory antrum, shaved off the cortex of the mastoid process and then allowed colored water to flow into the middle ear. The fluid came out through the retrofacial group of cells and cells of the tip, but did not invade the mastoid cells proper. When he removed the material with which he shut off the mastoid antrum and then made an injection, all the cells showed the colored fluid. This furnishes an explanation of the fact that infections of the middle ear that follow the retrofacial group of cells invade the tip, and provides an answer to the questions that have been propounded. Dr. Meltzer has done a fine piece of work and has made a definite contribution toward more efficient operations on the mastoid.

Dr. Philip E. Meltzer, Boston: In answer to Dr. Mosher, I did not intend to modify the nomenclature. I used my own classification to simplify the description of the cellular arrangement. One cannot improve on Neumann's classification. The arrangement of the cells may be self-evident to otologists who have had great experience in dissection of the mastoid, but there are a great many who do not have the opportunity to dissect temporal bones, and I believe that this description will be of value to them. I repeat, the roentgenogram is not reliable unless it is a stereoroentgenogram. Many factors make it difficult to obtain a good stereoroentgenogram, and not every one can make use of it when it is taken. Whether the cells cause the lateral sinus to take the position it does or the other way around is not determinable so far as I know. Dr. Babbitt asked if the lateral sinus determines the pneumatization. I think that it has nothing to do with pneumatization. My views in regard to pneumatization are those of

Wittmaack, Albrecht and others. Wittmaack's theory is fundamental, but I believe that there are other factors which influence the pneumatization. Dr. Babbitt asked whether the size of the sinus made any difference. I have never found that it made any difference whether the sinus was large or small. Development of the tip of the mastoid is due to the pull of the sternocleidomastoid muscle. Dr. Kopetzky's point is new to me, and I shall attempt to duplicate this experiment.

MICROSCOPIC ANATOMY OF THE EUSTACHIAN TUBE. MISS DOROTHY WOLFF, St. Louis.

In the embryo the epithelium of the eustachian tube differentiates later than lingual and nasal epithelium. In the third fetal month the tube presents a narrowing in the middle of its course. In the fourth month cartilage first appears lateral to the tube, near its pharyngeal end. At birth the tube is cartilaginous at its tympanic end. This condition was also found at 10 days of age. By 2½ months the bony tube is well developed. In the infant of 6 months a well defined isthmus was observed, the lumen being 2 mm. in height and 0.32 mm. in width. This width could conceivably have been distended 3 mm. before bony wall was encountered. A tubal tonsil (lymphoid nodule) may occur in the pharyngeal end of the tube. A normal adult tube at the isthmus measured 5 mm. in height and 1 mm. in width, expanding to 1.5 mm. inferiorly. This lumen could not have been distended because of the surrounding bony wall. (The isthmus is considered as the first point where the cartilaginous tube appears.)

Accessory cartilages were found to be really a part of the main lateral cartilage. The cellulae tubariae may be as large as, or larger than, the main lumen. They do not normally communicate with the main tube but are diverticuli of the tympanum. In pathologic cases, however, suppuration may break through from a diseased tube into these cells. The infection may pass forward into the petrous apex. The lumen of these pneumatic cells may then become lined with ciliated epithelium.

Pathologically, the tube may develop polypus-like growths at the tympanic orifice. These may become infected. Villus-like processes of the mucosa may develop and protrude down the lumen of the tube. These may be infected. In a case of stricture of the tube the number of glands near the pharyngeal end was greatly reduced. The connective tissue of the submucosa was more dense and fibrous than normal. The lumen was obliterated at a point 2 mm. below the isthmus, where it was widely patent. The epithelium at the stricture was neither columnar nor ciliated but was flat and in areas showed complete atrophy and obliteration.

All animal tubes observed were widely patent throughout their course.

DISCUSSION

DR. STACY R. GUILD, Baltimore: I should like to know the age of the patient in whom complete stricture occurred.

MISS DOROTHY WOLFF: Fifty-one years.

DR. STACY R. GUILD, Baltimore: Those who have not attempted to remove at autopsy the entire eustachian tube together with the middle and inner ear cannot appreciate one of the fundamental difficulties in obtaining the material that Miss Wolff has shown here.

DR. ARTHUR B. DUEL, New York: There is a tremendous amount of work involved in making the specimens and beautifully built models which make this presentation so illuminating. The wonderful anatomy of the tube makes one feel how crude one's therapeutic efforts are. Another point is the possibility that the tube is a source of infection of the petrous tip. Otologists should take notice of the anatomic relationship which Miss Wolff has indicated in her slides.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY

Jan. 18, 1933

BENJAMIN PARISH, M.D., *Chairman*

ABSCESS OF THE FRONTAL LOBE (MULTIPLE) SECONDARY TO FRONTAL SINUSITIS: REPORT OF A CASE. DR. WILLIAM GORDON.

The high mortality from abscess of the frontal lobe, in spite of the comparative rarity of the condition, merits a thorough study of the subject. Eagleton placed the mortality between 75 and 80 per cent. The literature was studied in an attempt to determine the reasons for the high mortality and to analyze the cases of the patients who recovered. The general phases considered were (1) etiology, (2) diagnosis, (3) anatomic relations and (4) treatment. In addition, an illustrative case is reported.

Etiology.—Infection: It is interesting to note that with so much nasal suppuration and so little aural suppuration, otitic abscesses are many times more numerous than those of nasal origin (900 abscesses of the middle fossa and 150 of the frontal lobe). The explanation of this offered by Eagleton is that the nose and the accessory sinuses are essentially a mechanism for protection not only of the upper respiratory tract but of the underlying bone of the nose and paranasal sinuses and the brain, whereas the mucous membrane of the mastoid antrum and cells is merely a cellular covering of the bone and is easily destroyed, allowing invasion of the bone. The mucous membrane of the nose and paranasal sinuses is for the purpose of removing infection or preventing a deeper ingress of infection. Should this fail, the elastic lamina propria, with its firm adhesion to the bone, prevents the micro-organisms from attacking the bone. When the protective mechanism of the nose is overcome, the protective mechanism of the brain tends to limit the infection by producing a pachymeningitis. Sinus infection, especially infection of the frontal sinus, is an important etiologic factor in abscesses of the frontal lobe. Myerson pointed out that every opening in the skull through which nerves or blood vessels pass constitutes a potential pathway for infection to the cranial cavity, and that bacteria are able to pass through some openings more readily than others.

Eagleton, in 1922, reported 150 cases of abscess of the frontal lobe. Sixty-four of these took their origin from the frontal sinus, and Ross H. Skillern and George M. Coates added 27 cases of this type, making a total of 91.

Trauma: When present, trauma is an important contributing factor in the production of abscesses of the frontal lobe—being more important here, perhaps, than in any other part of the brain because of anatomic relations. It occurred in 3 of the 27 cases in the Skillern-Coates series.

The traumatized brain is very susceptible to infection, and micro-organisms gain a foothold quickly. A certain amount of shock takes place in the brain cells, adding further insult to an already disorganized and devitalized structure. Eagleton says that while the frontal lobe is able to bear trauma well, it is very susceptible to a combination of trauma and infection.

Diagnosis.—All authorities are of the opinion that diagnosis is of utmost importance and that the earlier it is made, the better. Jason Dixon stated that no organ in the human body can conceal its trouble as thoroughly as a brain which harbors an abscess. Sonnenschein described how Eagleton laid special emphasis on a detailed history, placing before himself a calendar and noting on it the dates when certain symptoms occurred, especially vomiting, chills, headaches and vertigo. Barnhill stressed a careful history and repeated physical examination of the head for foci of infection, with careful searching of the ears and sinuses. He stated that an abscess of the brain never occurs spontaneously but results from infection.

Whenever a definite focus of suppuration is known to exist in the ear, nose, throat or lungs and the case presents cerebral symptoms one must be on the

lookout for an abscess of the brain. The specific symptoms and signs to look for are: 1. An initial vague chill arises with infection of brain tissue. By ascertaining the date of the chill, much information can be obtained about the formation of the abscess and its duration and virulence. 2. Headache, in the opinion of Eagleton, is always present. It may be dull or severe. It is unlike any other form of headache, except possibly that caused by a tumor of the brain. It is unrelieved by medication, is constant and tends to become progressively worse. 3. Vomiting is often induced by meningeal or cerebral suppuration. 4. General malaise and slow cerebration are sometimes present. 5. A slow pulse is a distinct symptom of cerebral compression and often may be associated with retinal changes. One may see a drop in the pulse rate followed by a return to normal—Macewen's sign. This dip is probably due to acute edema, the pulse rate returning to normal when the edema subsides and compensation is established. As stated by Coates, if the temperature and pulse rate were taken hourly or every two hours, Macewen's sign would be reported in a larger percentage of cases. 6. The body temperature is as a rule normal, subnormal or only slightly elevated. Eagleton enumerated the following factors: interference with the heat-regulating mechanism of the brain by cerebral suppuration; stimulation of the vasomotor center, and secretions from the brain that play a rôle in controlling temperature. 7. Papilledema is present, as a rule, when there is intense cerebral compression. It is usually absent in mild or moderately severe compression. An ophthalmologic examination is a sine qua non in all cases in which an abscess of the brain is suspected. 8. The cerebrospinal fluid often shows a mild increase in the cell count —between 100 and 200 cells. 9. Convulsions occurring in the presence of a suppurative focus in the nose indicate intracranial extension of that focus. 10. Coma is the terminal stage of compression. Any operative or expectant procedure during this stage is usually futile.

Anatomic Relations.—The meningeal barrier must be passed before the brain tissue is attacked (septic emboli excluded). Other protective mechanisms are the compact bony structure of the roof of the orbit and of the ethmoid, sphenoid and inner plates of the frontal sinuses. These plates of bone offer resistance to infection. Cerebral tissue, on the other hand, is the most highly specialized tissue in the body, and damage to it is irreparable. Eagleton stated that an intimate vascular relationship exists between the frontal bone and the dural covering of the frontal lobe. Zwillinger, after injecting colored fluid into the perimeningeal lymph channels, observed it flowing in the lymphatic channels of the mucous membrane of the frontal sinus. On an anatomic basis, therefore, the path of infection from a frontal sinus to the brain would be as follows: (*a*) by continuity of tissue; (*b*) by infection along veins—retrograde thrombophlebitis; (*c*) by a combination of the two; (*d*) by the perivascular lymph spaces, or (*e*) by infection of the diploic bone.

Treatment.—The rhinologist has one method of approach, namely, to open an abscess of the frontal lobe through the infected area. He cleans out the infected frontal sinus and then follows the path that the pathologic process has taken from the sinus to the brain, in order to provide drainage. T. H. Weisenburg, Francis C. Grant and others concur in this procedure.

Regarding methods of drainage, it is interesting to note the divergent opinions of authorities. King stated that none of the various forms of drainage materials produces adequate drainage in the true sense. He expressed the opinion that the most logical drain is the conical wire mesh of Mosher for the reason that the abscess cavity "plasters" itself against the drain and then herniates through the opening, thus effecting a cure. His method is to use no drain but to allow complete herniation of the abscess after making a large cranial defect over the abscess cavity. Four patients, including 1 with an abscess of the frontal lobe, recovered. He stated that no one procedure will cure all abscesses of the brain, nor will a combination of all procedures do so. The method to use depends on the type of case.

Dandy simply taps the abscess by introducing a ventricular needle, leaving it in place till the pus ceases to drip. Lemaitre urged that the surgeon respect the preformed adhesions in the subarachnoid space if they are present or produce adhesions there if none exist. He uses a Pravaz needle pushed through the meninges into the brain at the supposed site of the collection. When a drop of pus escapes, the needle is replaced by a groove director, and later a drain is left in situ for from twenty-four to forty-eight hours. Larger drains are gently inserted daily or every other day. He had five cases of cerebral abscess of otitic origin and one abscess of the frontal lobe, and all of the patients recovered. Cahill cautioned the surgeon to value the protecting meningeal adhesions.

Eagleton prefers an osteoplastic flap. Frazier and Grant in their recent series of 51 cases of abscess of the brain, of which 21 were abscesses of the frontal lobe, had 8 recoveries. Their technic is to wait until encapsulation of the abscess has taken place, about six weeks from the onset of symptoms. Rubber drains are then inserted and left in situ until the brain pushes them out. Coates seems favorably inclined toward drainage with iodoform gauze. His aim is to avoid trauma to cerebral tissue and to respect meningeal adhesions.

Report of a Case.—A boy, aged 11 years, three years before admission to the Graduate Hospital of the University of Pennsylvania, was hit over the right frontal sinus with a baseball bat. There was no loss of consciousness at that time, and the laceration was sutured by his physician. Three months before admission he had sharp pain in the head, followed by swelling in the region of the right eye and forehead. The area was incised, and his condition improved. Two months before admission, headaches recurred, accompanied by vomiting. A refractive error was corrected but without improvement. Medicine for the gastric disturbance relieved the pain. One week before admission to Dr. G. M. Coates' service the temperature was 98.8 F., the pulse rate, 110, and the respiratory rate, 20. Neurologic and general examination gave negative results. At 4 p. m. on the day of admission, the pulse rate fell to 64 (Macewen's sign). Examination of the eyes showed papillitis with edema rather than papilledema. The visual fields showed concentric contraction. Examination of the blood showed 11,500 leukocytes, with 78 per cent polymorphonuclears. Bárány's tests showed poor movements of the pelvic girdle and persistent past pointing, of considerably longer duration with the left arm than with the right. There were no shock responses and exaggerated nystagmus on rotation. Roentgen examination showed an old suppurative sinusitis of the right frontal sinus with erosion of the anterior and posterior walls and also of the anterior portion of the floor of the sinus. A diagnosis of epidural abscess was offered.

At the preliminary operation both frontal sinuses were opened, and communication was established between them. The left sinus showed polyps and hyperplastic tissue. The right was small but showed a necrotic posterior wall, which after removal exposed a dura under tension which presented a yellowish necrotic spot 1 inch (2.5 cm.) to the right of the midline and ½ inch (1.3 cm.) from the floor of the anterior fossa. A coffer-dam of iodoform gauze was inserted at the edges of the bone surrounding the exposed dura, and the wound was dressed, the right frontal sinus being left practically open.

On the following day, the patient was again operated on. The coffer-dam was removed, and the wound irrigated with a 1:5,000 solution of corrosive mercuric chloride. The tension of the dura appeared increased, particularly near the midline. The dura was incised at the yellowish spot previously noted. There was no subdural abscess. A brain cannula was inserted to the extent of 1 inch, and much resistance was encountered. Evidently a capsule was present. The cannula was inserted after the capsule was incised with a long bistoury. One drachm (3.7 cc.) of pus was recovered from the abscess cavity. The tension toward the midline persisted, so another incision and tap were made in the tense area. About 4 drachms (15 cc.) of pus under tension was obtained. Both incisions were enlarged so that they became one, and the cavity was packed with iodoform gauze. The skin flap was left fairly wide open.

The patient's condition was satisfactory for a week. At the end of that time, severe headache in the frontal and occipital regions developed. Examination of the eyes showed definite papilledema with irregular flattening of the visual fields, particularly of the right eye. The temperature began to rise, and the pulse rate showed a dip to 62 (Macewen's sign). Early one morning he had a convulsion. He became incontinent and showed a positive Babinski sign on the right side, and his mental condition, which had been clear, became dull and listless. He was rushed to the operating room. A large mass of strangulated and necrotic brain tissue was seen protruding from the wound left by the previous operation. On removal of this tissue, the mass was found to contain an abscess which protruded with the hernia. From the original deep abscess cavity in the external side of the frontal lobe, clear fluid dripped. This was presumably cerebrospinal fluid and suggested rupture into the lateral ventricle, probably during the convulsions that produced the hernia.

At autopsy, the abscess of the frontal lobe was found to extend into the right lateral ventricle; this opening was large enough to admit a finger. There was no evidence of meningitis on the upper or lower surface of the cerebrum. When the tentorium was opened in order to remove the cerebellum, a large quantity of pus was found bathing the entire outer surface of the cerebellum and extending down the posterior aspect of the brain stem.

DISCUSSION

Dr. George M. Coates: In all cases of infection of the sinuses as well as of the ears, the persistence of headache should lead one to suspect an intracranial lesion. In one or two cases of our series, headache was the only symptom noted until late in the disease. Dr. Gordon says that these cases should be diagnosed as early as possible, but I do not believe that one should necessarily operate early. Last week, in Boston, a paper on abscess of the brain was read in which operation was advocated as early as the diagnosis could be made. What was meant was that operation should be performed as early as possible in cases showing symptoms of abscess of the brain. It seems to me that the patient has a better chance of recovery if the infection in the brain becomes walled off and local resistance is established.

The type of drainage may be variable, but I have found iodoform gauze successful. In Dr. Gordon's case a coffer-dam made of gauze was placed between the bone and dura in order to create pressure and so promote adhesions between the dura and the brain. This walling off, unless nature has already accomplished it, is essential to prevent the escape of cerebrospinal fluid and the spread of septic meningitis when the abscess is opened. It is interesting to note that in Boston diathermy is used for this purpose.

A history of trauma is interesting in abscess of the frontal lobe for almost all patients give a history of trauma at some time—not necessarily recent but often old trauma. Trauma seems to render this area of the brain susceptible to infection.

When there are symptoms of abscess of the brain in the presence of suppuration of the ethmoid or frontal sinus, and one can rule out an aural infection, I believe that the approach should be through the frontal sinus. Any abscess of the frontal lobe, no matter from what location the cause may come, is best reached through the frontal sinus.

Dr. Benjamin Shuster: Macewen's sign, consisting of a dip in the pulse rate, is significant of abscess of the brain, particularly when accompanied by a rise in temperature. Every time I have a patient with mastoiditis or sinusitis and the temperature goes up and the pulse rate goes down, I get a chill. Headache and vomiting together, as Gower stated, are a sure sign of an intracranial lesion. Moreover, headache is always unusual in children.

The results of vestibular tests in this case were negative. I tested the patient a great many times and did not get a response. These patients are usually susceptible to testing and are disturbed by it. Weakness of the left arm was

significant in connection with the finding of the abscess of the right frontal lobe, and the responses of the pelvic girdle suggested a lesion of the vermis. It is usually a great help in these cases to think neurologically. The reason for the high mortality is usually the presence of meningitis.

INTRA-ORAL DRAINAGE IN SUBLINGUAL INFECTION (LUDWIG'S ANGINA). DR. KARL M. HOUSER.

Almost one hundred years ago (1836) Ludwig published his observations on the oral inflammation that later was to bear his name. There was much debate from time to time regarding the propriety of considering the disease an entity, but early in the twentieth century the publications in America by Davis, Thomas and Price aided greatly in establishing the present conception of the disease, so that Ludwig's angina has practically universal acceptance.

Cases that fill the requirements for this diagnosis are not common. Over a period of eight years, the general surgical service at the Hospital of the University of Pennsylvania had but 12 patients with this disease in a total of 18,705, or a percentage of 0.07. This total did not include the patients admitted to the gynecological, ophthalmologic or otolaryngologic services. Three additional patients were treated at the department of otolaryngology. The disease is most frequent in children and in young adults, and males are more often affected than females. Fifty-five per cent of all cases occur in the first three decades of life, although at no age do persons seem to be immune. Fetterolf concisely referred to the condition as "a phlegmon in the floor of the mouth above the mylo-hyoid muscle." Definitions all agree that the sublingual space is involved in true Ludwig's angina. In the earliest stages, the disease is confined to the floor of the mouth, submandibular involvement being a later manifestation. Blassingame concluded, after injecting ink into various parts of the mouth and throat of dogs, that infection from the posterior teeth or from that part of the jaw may: (1) extend into the sublingual space and cause Ludwig's angina; (2) pass through this space to the deep cervical nodes, or (3) involve both of these areas. The tonsils and the anterior teeth are not likely to be causative factors. Ashhurst emphasized the fact that this condition is cellulitis and not lymphadenitis. In 11 of 18 cases reported by him there was a definite history of dental caries, and in only 3 of the 7 remaining cases could dental caries be absolutely excluded.

Anatomically, the sublingual space is bounded laterally and in front by the body of the mandible. Below is a firm diaphragm made up of the two mylohyoid muscles fused in a median raphe attached laterally and in front to the inner surface of the mandible and posteriorly to the hyoid bone. These muscles are enclosed in a definite and fairly dense sheath, which near their bony attachment thins out in a firm, unyielding aponeurosis. External to this muscle is the deep cervical fascia, which, although rather thin, is strong and unyielding. The fascia is attached to the inferior edge of the mandible and splits to envelop the anterior bellies of the digastric muscles, all of which gives additional firmness to the floor of the mouth. From the posterior borders of the mylohyoid muscles are continued the pharyngeal constrictors, separating the pharynx from the neck. A deficiency exists between the middle constrictors and the mylohyoids, giving rise to an opening extending laterally from the hyoid bone to the inner side of the mandible near the angle. This gap is principally filled by the hyoglossus muscle and the curve of the horseshoe-shaped submaxillary salivary gland. The posterior boundaries of the sublingual space are less easily visualized but are just as definite. As the tongue dips downward and backward toward the epiglottis, the free edges found farther forward in its more motile portion no longer exist. The palatoglossus and the more robust styloglossus have attached themselves at each side and constitute a firm, mucous membrane-covered, muscular ridge. The superior boundaries of this space are simply the tongue and the mucosa of the floor of the mouth.

When one takes these details into consideration, it does not seem strange that Ludwig's angina causes a boardlike hardness under the mandible on account of

the tenseness of the mylohyoid muscles and the deep cervical fascia, and that it causes swelling of the floor of the mouth which pushes the tongue up and back. When one realizes that the distance from the symphysis of the jaw bone to the base of the epiglottis in the adult is but 2½ inches (6.27 cm.), it is not remarkable that edema of the larynx follows an unrelieved condition of this sort. Nor is it strange that pus is often found in the neck in the region of the superficial part of the submaxillary salivary gland when a definite anatomic communication exists between this region and the site of the initial infection.

It is remarkable, however, that more attention has not been given to the relief of this condition by intra-oral operation early in its development. In the large series of cases reported by Thomas, there were 17 cases of spontaneous evacuation. All of the abscesses opened internally; in 1 there were both an internal and an external opening. Therefore, it seems that when spontaneous opening is possible nature chooses the internal route. If the mouth cannot be opened, as is often the case late in the disease, or if pus is already present in the neck, external opening only will suffice. But if the patient is seen early, there is a possibility of bringing the infection to a speedy termination with relatively simple intra-oral surgical maneuvers. If this fails, no harm has been done, and the external operation can always be performed.

Three cases were reported.

CASE 1.—A young man presented sublingual swelling on the right side, elevation of temperature, leukocytosis, dysphagia and boardlike hardness of the tissues under the chin and the right mandible. Spontaneous rupture of a sublingual abscess occurred through an opening near the right side of the base of the tongue.

CASE 2.—A man, aged 42, complained of a tickling sensation in the right side of his throat for four days. Two days later he showed sublingual swelling on the right side. The tongue was pushed toward the roof of the mouth and to the left. There were marked dysphagia, slight dyspnea and a temperature of 101 F. Through an incision over the swelling several tablespoonfuls of pus was evacuated.

CASE 3.—A boy, aged 13, with symptoms of sore throat and dysphagia which had grown progressively worse for six days showed swelling of the tongue and the sublingual tissues. The tissues below the chin were hard and swollen, and the right submaxillary salivary gland and lymph nodes were swollen and tender. After a temporary improvement of symptoms the patient rapidly became worse. Through a small incision, enlarged with a hemostat, about 2 ounces (60 cc.) of foul pus was evacuated. A rubber dam was inserted, and further evacuation was accomplished. The patients in all 3 cases made rapid recoveries.

DISCUSSION

DR. BENJAMIN PARRISH: What guides Dr. Houser in determining the time for making the incisions?

DR. GEORGE M. COATES: Does Dr. Houser think that Ludwig's angina is always due to dental caries. I saw a case in which there was definitely a peritonsillar abscess. I saw as consultant another case in which there was a peritonsillar abscess and the patient suddenly took a turn for the better. Could Ludwig's angina be due to peritonsillar abscess?

DR. WARREN DAVIS: I had a case associated with an abscess around a large stone in the sublingual duct in which the patient was relieved after intra-oral drainage and removal of the stone. At the Jefferson Hospital, the rule is to try intra-oral drainage unless pus has already formed outside the mylohyoid muscle.

DR. KARL M. HOUSER: The time of incision can be judged by the symptoms and by bimanual palpation with one finger in the mouth and one outside. If the swelling is fairly well marked and the patient obviously sick, one may just as well try an intra-oral incision.

In regard to etiology, I can say nothing except that dental caries is not always but usually given as the cause (in about 85 per cent of cases).

MASKED MASTOIDITIS. DR. WILLIAM HEWSON.

In referring to mastoiditis as masked, one merely cloaks one's ignorance. The forms of this kind of mastoiditis are so numerous that it was not practical in this report to speak of them all. Dr. Hewson and Dr. Eiman called attention to three cases which should put one on guard in considering this very masked subject. The case that Dr. Hewson reported was one of mastoiditis which became clinically recognizable after meningitis and coincident with pneumococcic septicemia. The child got well, and on reviewing the history this sequence seems the logical one.

Billy P., 5 years old, was admitted to the Abington Memorial Hospital on Jan. 1, 1932, complaining of pain in the back, stiff neck and loss of memory for events of the day before. During the previous night he had been restless and twitching and had vomited several times. He had returned home two days before after a stay of a week in the Northwestern Hospital, where he complained of pain in the back of the head and was treated for sinusitis. His mother told of his having generalized convulsions at the age of 1 year; both ear drums were opened, and pus drained for a long time. During his second year he continued to be sickly and frequently had "sore ears." He then had his tonsils removed but continued to have frequent colds. In November, 1931, after an acute cold some swelling developed about his right eye and he was taken to the Northwestern Hospital.

Examination on admission to the Abington Memorial Hospital showed the patient to be irritable when touched; if let alone he would lie with his head drawn back and his eyes closed, breathing quietly. He looked sick, out of proportion to his temperature of 102.4 F. The right upper eyelid was slightly swollen, and the conjunctiva was red; he dreaded the light. The left eye was held tightly closed, though apparently it was not inflamed. Examination of the eyes otherwise gave negative results. The ears were normal, and there was no tenderness about the mastoids. The nose showed no discharge. There was some deviation of the septum constricting the right nostril, and the mucous membrane was only moderately congested. There was slight weakness in the muscles of the right side of his face. The tongue protruded normally in the midline. The neck showed no adenopathy but was rigid. The knee jerks and the abdominal and other reflexes were exaggerated. There was a bilateral Kernig sign, and the Babinski reflex showed slight flexing of the right little toe. There was slight weakness of the right arm and leg. Spinal puncture yielded a fluid under a pressure of 40 mm. of mercury and containing 540 polymorphonuclear cells. The leukocyte count was 23,650.

From 3 o'clock until 8 or 9 in the evening, the condition remained about the same; so with the encouragement of the pediatric service, under whose care the patient was, it was determined to improve drainage from the maxillary and posterior ethmoid sinuses. Parts of both middle turbinates were removed, and an entrance was made into the maxillary sinuses and a few posterior ethmoid cells. No pus or abnormal secretion was found. With a faint hope that the ears might yield some evidence, both drums were incised, but no discharge was obtained. The pediatric department continued to observe the patient and could find no focus demonstrable in the lungs or in the gastro-intestinal or the genito-urinary tract.

On the second day after operation (January 3), Dr. George Wilson examined the patient and agreed to the diagnosis of meningitis of undetermined type. He also suggested that an abscess of the brain or cavernous sinus thrombosis was to be watched for. The nose showed a mucopurulent discharge, but the ear drums had healed without discharge. A culture of the spinal fluid was negative. The temperature, pulse rate and respiratory rate remained about the same. On January 4, the patient seemed somewhat more toxic. The right ear drum showed some inflammation in the upper posterior quadrant with slight bulging, and so was incised. There was some tenderness over the right mastoid antrum. The left ear drum lacked a little in luster but was otherwise normal. The next day, the left ear drum ruptured, and some serosanguineous fluid was discharged. The general condition seemed improved, the meningeal symptoms gradually subsiding. The

results of roentgen examinations during the next two days corresponded with the clinical finding of bilateral mastoiditis, developing first on the right and soon afterward on the left side. On January 7, both mastoids were opened and found to be of the diffuse large cellular type. Destruction of bone was scant and confined to the region of the mastoid antrums. Pus was found in abundance in the antrum and the tip of each mastoid. Large cells with normal-appearing mucosa were found overlying the sinus plates and on the left side posterior to the knee of the sinus. Parts of the dural plate and the sinus plate were removed on the right side, exposing a healthy-looking dura and sinus.

In the next two weeks, the wounds in both mastoids healed completely. The patient continued to have a low degree of fever, however, and the blood count showed cellular anemia with the presence of a few myelocytes and Metz forms. Transfusions improved this condition considerably, but by February 1 the patient began to have septic elevations of temperature and headache in the left temporal and left occipital regions. On February 6, the left mastoid was reopened and the sinus plate removed for about ¾ inch (2 cm.). No pus was found anywhere, but unhealthy granulations were removed from the region of the aditus. The lateral sinus showed localized thickening and inflammation, with adhesions to the sinus plate. The sinus was patulous and appeared healthy above and below this area, and so was not incised. Recovery was more certain after this operation, though the course was stormy and blood cultures, formerly negative, at this point showed Pneumococcus type 3. The ultimate recovery was attributed to autogenous vaccine therapy.

The correct interpretation of this case hinges on the meningitis. This was undoubtedly of toxic origin because bacteria were not found in the spinal fluid. Causes other than an infection of the ears, nose or throat can be ruled out, though the child was of a neurotic temperament and, according to the report of the Northwestern Hospital, frequently had spasms before micturation. Consideration may be narrowed to the sinuses and ears. The recent history suggests sinus infection, though this was not borne out by examination at either the Northwestern or the Abington Memorial Hospital. The swelling about the right eye might have been accounted for by the conjunctivitis. The reports of roentgen examination were not definite enough to be of much help. If there had been a sinus infection sufficient to cause the meningitis, it is reasonable to suppose that after the intranasal operation this would have been manifested by a continued purulent discharge. No effort was made to eradicate sinus disease but merely to afford drainage. The mucopurulent discharge that developed cleared up in a few days. If a focus of infection in the nose had been responsible for the virulent mastoiditis, some more virulent infection would have appeared first in the nose.

According to the report of the Northwestern Hospital on the patient's admission in December, 1927 (when he was 1 year old), he had fever and convulsions. A diagnosis of acute otitis media and bronchitis was made at that time. Myringotomy was performed, and thick pus discharged profusely from both ears for several weeks. Because of the large cellular type of mastoid found at operation, it is conceivable that some residual infection might have persisted in an attic cell or beneath the tegmen. Absorption from this area might have caused the toxic or serous meningitis. When myringotomy was performed later, conditions became changed in the middle ears and antrums, which favored the growth of the more or less quiescent pneumococcus in the direction of the mastoid cells. The meningeal absorption therefore stopped, and the mastoid cells became receptacles. The virulence of the infection can be attested to by the rapidity with which localized sinus phlebitis and septicemia developed. If meningitis had followed the acute mastoiditis, it would have been easy to suppose the course of infection, but since meningitis developed first, this may be considered a case of masked mastoiditis.

MASKED MASTOIDSTIS. DR. JOHN EIMAN.

It has been my fortune (good or bad) to perform autopsies on children whose ears had been carefully examined by competent aurists and pro-

nounced normal and who, however, at autopsy, showed pus in the tympanum and mastoid and signs of mastoid complications. I wish to add two cases to the one reported by Dr. Hewson. Though I do not wish to detract anything from his ability and skill, these cases illustrate that the most careful otologist may be mistaken. These cases occurred at the Abington Memorial Hospital within a month, at a time when every one was suspecting aural infection on the slightest provocation.

The first case was that of a girl, 6½ years old, in Dr. Coates' service, who was admitted with the history of headache, cough, vomiting and slight fever developing two weeks before admission. Her physician suggested that she had intestinal influenza. The abdomen became bloated; the stools were green and slimy, and later she was constipated. Her cough continued to be loose and productive, and headache was severe when she sat up. Her mother said that she had been weak, nervous and fidgety and had a distressed expression almost constantly. Her general health had been fair and, except for attacks of colic and for pneumonia at the age of 3 years, she had had no severe illness until the present trouble began.

Examination showed a thin, pale girl, whining and crying with a shrill voice. The neck was rigid, and the head was drawn back. The eyes were staring, but she dreaded the light. The pupils were dilated and reacted sluggishly to light. The ears contained large plugs of inspissated wax; after these were removed, the drums looked normal. Spinal puncture yielded a thick turbid fluid under a pressure of 18 mm. Cisternal puncture, two days later, showed the same kind of purulent fluid. The white blood cell count was 38,000, with 87 per cent polymorphonuclears. Spinal fluid cultures showed beta hemolytic streptococci and from 15,000 to 20,000 cells per cubic millimeter, with 1,005 polymorphonuclears.

The patient's condition grew progressively worse, and in spite of cisternal and spinal lavage, blood transfusions and the use of an antipneumococcus serum, she died on the eighth day after admission.

At autopsy, the base of the brain showed a plastic creamy exudate with a greenish tinge over the entire base, but especially thick in the region of the pons. The cerebral vessels were injected and the convolutions somewhat ironed out. Both lateral ventricles were distended with purulent material. The spinal cord in the thoracic region showed some thick exudate, and the subarachnoid space contained serosanguineous fluid. The dura was not thickened, and there was no thrombosis of the venous sinuses. The right ear showed no thickening or injection of the tympanic membrane, but there was a slight amount of pus in the middle ear and in the mastoid cells. The left ear showed slight redness and thickening of the tympanic membrane, and there was pus in the mastoid cells. The ethmoid sinuses were clear.

The second case was that of a girl, aged 12 months, who was admitted to the hospital on account of convulsions which followed a period of five days of vomiting after every feeding. On the day of admission, she became rigid and her jaws could not be opened. Later, there were twitching of the left arm and frothing at the mouth. Spasticity would relax only to return. There was no history of injury or of infection. She was one of twins but had been strong and in good health, except for an occasional gastro-intestinal upset. She had no history of respiratory infection.

Examination gave negative results so far as the heart, lungs, nose, throat and ears were concerned. She was comatose and lay with the eyes partly closed. The eyeballs remained fixed and in the right lower corner. The left angle of the mouth twitched constantly, and the jaws were spastic. The neck was easily flexed, and Brudzinski's sign was negative. All four extremities were extended and spastic. The left arm was in constant clonic spasm. There was bilateral ankle clonus. The abdomen was distended and tympanitic. She died, about ten hours after admission, having a temperature of 105 F.

Spinal tap after death showed a yellowish fluid. Autopsy showed an organized clot in the posterior part of the superior longitudinal sinus. The veins emptying

into this sinus were markedly distended and remained so after removal. All the other nervous sinuses were normal except the torcular herophili, which contained organized clots. The nasal accessory sinuses were opened and found normal. Both mastoids and middle ears showed pus which on culture yielded Streptococcus, Staphylococcus and Bacillus coli. Blood culture showed beta hemolytic streptococci.

In both of these cases the origin of the fatal illness was probably an unrecognized mastoid infection. This is more easily traced and understood in the first case, in which meningitis prevailed, but in the second case I am at a loss to explain it.

DISCUSSION

DR. ADDINELL HEWSON: These three cases present interesting problems clinically, pathologically and anatomically. The first two reported by Drs. Hewson and Eiman illustrate the relationship between the middle ear and the meninges, while the third case particularly demands an understanding of the venous anastomoses. I shall not attempt a clinical or pathologic consideration, but I believe that an anatomic summary will be of interest at this time.

Concerning the transmission of infection through the mastoid, three principal routes may be considered: 1. In babies up to 1 year old, nonunion between various centers of ossification affords ingress through vessels and channels which later become blocked by union of the bony sutures. The squamous, mastoid and petrous portions of the temporal bone afford an example of this. 2. Normal dehiscences exist in places such as the openings for passage of nerves, blood vessels and lymphatics, of which the ductus endolymphaticus and the aqueduct of the vestibule are good examples. 3. Abnormal dehiscences may occur almost anywhere; a frequent one in the temporal bone is beneath the superior petrosal sinus and overlying the superior semicircular canal. That infection may reach the middle ear and mastoid from the septic throat or nose is commonly recognized, but one is apt to lose sight of these numerous small passageways which can carry the trouble to the more vulnerable parts of the cranial cavity.

The venous circulation about the head seems to arrange itself into three systems: (1) the vessels outside of the cranial cavity, (2) those within the cranial bones and between these and the dura and (3) those inside the dura. The veins of the skull and face, as well as the internal maxillary vein, drain into the external jugular system. Those in the cranial bones and between these bones and the dura connect with the so-called emissary veins, emptying into the superior longitudinal sinus, the torcular herophili, the lateral sinus and the cavernous sinus. The veins inside the dura, or cerebral veins, empty into the superior longitudinal, cavernous and superior petrosal sinuses. The great cerebral vein (the "vein of Galen") from the interior of the brain empties into the straight sinus and thence into the torcular herophili.

From this it is evident that free communication is afforded between the cerebral, endosteal and superficial veins of the skull by way of the communicating veins through the large venous sinuses. The large and persistent lumens of these sinuses are assured by their attachment to bones and fibrous ligaments of the skull as well as by a diminished amount of elastic and muscle tissue in their walls. Circulation through these sinuses is consequently retarded and further impeded by pacchionian bodies and intrasinus trabeculi projecting into their lumens. Should infection reach the superior longitudinal sinus, as is presumed in Dr. Eiman's second case, thrombosis would probably be facilitated by the factors mentioned, causing slowing of the circulation of sinus blood.

Book Reviews

The Operative Story of Cleft Palate. By George Morris Dorrance, M.D., assisted by Enayat Shirazy, D.D.S. Price, $6.50. Pp. 564, with 534 illustrations. Philadelphia: W. B. Saunders Company, 1933.

In this volume the authors undertake to relate the complete story of operative correction of the cleft palate. How successful they have been in compiling the story may be surmised from the fact that the text contains 534 illustrations and there is appended a bibliography covering more than 80 pages. This unique surgical field is on the borderline encroached on by the oral surgeon, the otolaryngologist, the dentist and the general surgeon. A perusal of this story of operative work on the cleft palate will instil a wholesome respect for this field of surgery. The work impresses the reviewer as being complete and will be perused with profit by any one who would undertake this field of surgery.

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FOREIGN

Collegium Oto-Rhino-Laryngologicum Armicitiae Sacrum

President: Professor Gunnar Holmgren, Karolinska Mediko Kirurgiska Institut, Stockholm, Sweden.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg, 143, Groningen, The Netherlands.
Place: Stockholm, Sweden. Time: September 2-5, 1934.

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. W. P. Wherry, 107 S. 17th St., Omaha.
Secretary: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Place: Cleveland. Time: June 11-15, 1934.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
President-Elect: Dr. J. M. Wheeler, 30 W. 59th St., New York.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: 1934.

American Bronchoscopic Society

President: Dr. W. F. Zinn, Medical Arts Bldg., Baltimore.
Secretary: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia.

American Laryngological Association

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Secretary: Dr. W. V. Mullin, 9204 Euclid Ave., Cleveland.

American Laryngological, Rhinological and Otological Society

President: Dr. J. W. Jervey, 101 Church St., Greenville, S. C.
Secretary: Dr. R. L. Loughran, Bridgewater, Conn.
Place: Charleston, S. C. Time: April 3-5, 1934.

sections

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TEMPERATURE (Mean monthly—40-year average)		49.7	52.2	57.5	64.3	71.4	77.4	82.7	80.7	69.7	59.2	46.8	57.5	49.8
RAINFALL (In inches—40-year average)		.8	.96	.81	.32	.14	.23	.24	2.46	1.0	.6	.76	1.09	Total 11.57
HUMIDITY Monthly (40-year average)	A.M.	61	55	64	48	40	42	84	93	54	35	73	47	68	57	74	62	Aver. 49.5
	P.M.	35	32	27	22	42	26	15	9	17	7	36	73	57	29	02	65	41	18	29.9

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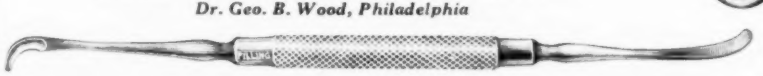

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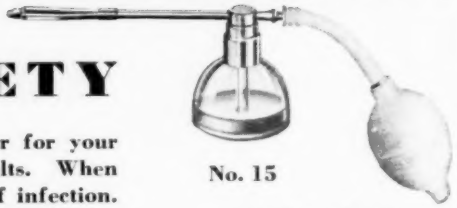

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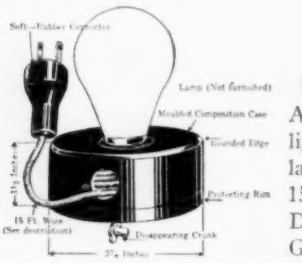

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